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THE MANAGEMENT OF SOME COMMON SKIN DISEASES BY THE GENERAL PRACTITIONER.*

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It is always difficult for the specialist to submit practical procedures which can be applied by the general physician. And this difficulty arises largely from the uncertainty as to the logical limits of the activities of the general practitioner.

The principal cause of this uncertainty is apparent: namely, that the present basis for fixing the boundaries both of general practice and of the various medical specialties is obviously founded upon practical considerations, and not upon exact scientific or logical division and classification. Pediatrics, for example, is bounded by the criterion of the age of the patient; endocrinology, by the effects of a special system of glands, characterized by the absence of ducts. The fields of the surgeon, of the roentgenologist, of the physical therapeutist—all are determined by the modalities employed; the spheres of the syphilologist and of the tuberculosis specialist include all manifestations attributable to the respective etiologic agents; the province of the venereologist embraces those diseases which are usually acquired through sexual exposures; and, to terminate an enumeration which would prove more tedious than profitable, the specific organ or part affected determines the domains of many specialists, such as those of the proctologist, the gastroenterologist, the ophthalmologist, the neurologist, and the dermatologist.

When one considers these various and entirely different bases for division and classification, there can be little wonder that the boundaries between the specialties are so frequently wanting in sharpness of definition. Nor can one be astonished that this lack of sharpness often leads to the vexing question as to where one specialist's activities should rightfully cease, and where another's should begin: a question which is, not uncommonly, highly and bitterly controversial, and which so often presents a problem inaccessible to satisfactory or accurate solution.

When, in the light of the foregoing, one considers the boundaries of the province of the general practitioner, one finds that the lines of demarcation are here not only the most vague and unsharp, but also the most inconstant and elastic. For most particularly the general practitioner's field is incessantly widened or contracted by many variables, such as the accessibility or lack of accessibility of various specialists, the bent and training and particular aptitude of the individual practitioner, the inclination and choice of the individual patient; and, perhaps unfortunately but most certainly, by the economic conditions of both physcian and patient. These points are so apparent that they scarcely merit mention. The general practitioner in an isolated rural community will necessarily have to treat a much greater variety of conditions than the city practitioner who can readily call upon an expert in every specialty. Similarly, the general practitioner established in a poorer community will be less able to call upon specialists; and in bad times or depressions (or even in recessions), both patient and physician will be less inclined to have recourse to a specialist. Because of these and other factors, one encounters every conceivable kind, character, and size of general practice. On the one extreme, there is the fashionable city practitioner who, if he called upon every available specialist at every opportunity, would soon shrink to the status of a mere routing- and shipping-clerk; and, on the other extreme, there is the doctor who is forced by circumstances to manage every presenting malady and situation, and who

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often becomes adept in the treatment of an astonishingly wide variety of diseases.

I have indulged in this rather lengthy preamble in order to demonstrate the dilemma of a speaker when confronted with a title containing the words ". . . in general practice". For, not only does the scope and therefore the interest of the general practitioner vary from individual to individual, but the field of even one and the same man may expand and contract with Alice-in-Wonderland-like rapidity under the influence of ever-changing circumstances and forces. But in spite of the inherent difficulties, I hope that I shall be able to select for our present discussion certain dermatoses in which the milder cases should be well within the province of every general practitioner.

For examples, both acne vulgaris and certain common eczematous dermatoses will inevitably be encountered in every general practice. For it is generally acknowledged that acne vulgaris and the eczematous dermatoses—including eczematous "ringworm" and so-called "athlete's foot"—today constitute the most common skin diseases. And it is unreasonable to suppose that early or mild forms of these skin affections will all be treated by specialists in dermatology, regardless of how wealthy the community or how accessible the specialist. Surely, the adolescent with a few occasional blackheads and "pimples", or the patient with a small patch of "eczema" or a slight scaly or itching eruption of the hands and feet will often necessarily be the problem of the general practitioner who, in many instances, may have been consulted primarily because of some other, non-dermatologic disturbance. It follows that, if the medical profession as a whole were somewhat better equipped to manage the innumerable mild cases of acne and of eczematous dermatitis, the public would be spared many of the present abuses. For, *if the general physician fails to exhibit interest in and knowledge of such skin conditions, it is inevitable that many of these mild cases will go—not to the dermatologist*—but *to some other source for information, for advice, and even for treatment.* And I believe that, not only the medical profession —including specialists and non-specialists—but also the majority of intelligent, unbiased laymen will agree that it is incontrovertible that even the mildest skin diseases *should be treated by physicians, and not by non-medical cosmeticians, beauticians, barbers, coiffeurs, masseurs and food faddists;* and that most patients, even with the mildest dermatoses should most emphatically *not be left to the mercies of the druggist or to the diagnostic and therapeutic clairvoyance of the manufacturers, distributors and advertisers, who vaunt the host of magic proprietary remedies, alkalinizers, blood "purifiers", laxatives, fungicides, "glamorous" cosmetics, vitamin and endocrine creams and soaps, and panacea patent medicines or branded foods.* Nevertheless, the great profits which may be made from the sale of such preparations are mainly dependent upon continuous advertising, and this extensive and persistent "high pressure" advertising has largely succeeded in befuddling and misleading the public, so that it seems to me possible that in this country today only the smaller number of sufferers from mild and common dermatoses is being treated by physicians; while the greater number is seeking relief in remedies and procedures which are often not based on scientific and established fact, but rest solely upon false claims and absurd superstitions. Such superstitions include, for example *the myth of "skin foods"; the saga of "getting out the poison"; the fantasy of "too rich blood"; and the fairy tale of "too much acid".*

All these self-evident evils can be controlled only if the family physician ceases to display indifference when his patients with colics or colds or "rheumatism" ask his advice regarding their complexions and acnes, or concerning their "eczemas" or "athlete's foot". And furthermore, it seems to me apparent that the physician who tries to help his adolescent patients over their acne, or the housewife over her eczema, will gain more confidence and esteem than his colleague, who shrugs his shoulders or turns his back.

I believe, therefore, that each general physician and every non-dermatologic practitioner should be familiar with the basic concepts of pathogenesis and mechanism of the most common skin affections; and should be proficient in at least a few of the correct, simple, modern measures for initiating the treatment of mild cases of certain of the most prevalent skin diseases, such as acne vulgaris and eczematous dermatitis both of fungous and of non-fungous origin.

ACNE VULGARIS

Acne vulgaris is undoubtedly in some way based upon the functions of the endocrine glands, and is

directly or indirectly connected with gonadal activity. The proofs of this statement are convincing, but far too numerous and lengthy for detailed mention here. (For discussion and bibliography on this subject, see, for example, Sulzberger, Rostenberg, Jr., and Sher, *New York State Journal of Medicine*, 34:1, 1934; J. Michael, *J.A.M.A.*, 105;327, 1935; and the leading article on therapy in The Year Book of Dermatology and Syphilology for 1933). Here I need cite only the facts that acne and baldness are practically unknown in eunuchs; that comedo-acne may be present shortly after birth when the infant's skin is still manifesting the effects of the maternal hormones; that acne vulgaris is absent during childhood and old age, and usually appears with the beginning of puberty and often disappears with the cessation of sexual development or of gonadal activity; that acne often exacerbates with the menses or with the new adjustments or maladjustments of menopause; that acne often exacerbates or disappears in pregnancy; and that the primary site of the acne lesion—the hair follicle and sebaceous gland (the so-called pilosebaceous apparatus)—is related to the mammary glands, the apocrine glands, and to those hair-producing organs which are well known to be highly susceptible to stimuli arising during puberty, stimuli which are in some way, directly or indirectly, connected with gonadal development and with the production of sex hormones.* But in spite of the recognition of the role of the endocrine glands in the production of acne vulgaris, no present method of endocrinologic therapy has proven itself to be generally useful in the treatment or in the prophylaxis of this dermatosis. I say this deliberately and advisedly, and in the face of the recent articles reporting the beneficial effects of the anterior pituitary-like and of the ovarian follicular hormones. While it is a fact that a few isolated cases of acne may be benefited by hormonal treatment, the majority of these patients are helped more by the proper and judicious use of local superficial X-ray, than by any other remedy or groups of remedies. And it is deplorable that even today sufferers from severe acne are often denied possible relief because of general ignorance of the fact that roentgen treatment, as practiced by the well-trained and conscientious dermatologist, offers a method of treatment without danger and with the greatest chances of success. I believe that this is the place for me to state that the therapeutic index of proper dermatologic roentgen therapy compares favorably with that of the majority of remedies employed in modern chemotherapy. It is, therefore, my conviction that, wherever strictly indicated, roentgen therapy must be administered, rather than to allow the disfigurement of an active acne to continue to embarrass and to embitter these patients, and, as is not infrequently the case in adolescents, even to annihilate their chances for success, both in business and in private life. For such acnes, even though resistant to other measures, will nevertheless often yield to a dosage of the proper type of roentgen ray which lies well within the limits of absolute safety. However, the administration of X-ray, in order to be both effective and entirely safe, requires not only the strictest selection of cases plus special knowledge and technic, but also demands special apparatus. As a rule, only the dermatologist is equipped to give the type of superficial radiation indicated in skin therapy; and I must emphasize that the apparatus and the experience of even the trained roentgenologist is not essentially suited for the best radiologic treatment of acne. Moreover, before proceeding to roentgen therapy, it is obligatory for the dermatologist to consider in differential diagnosis, and to exclude the many other dermatoses

*Among the many additional arguments which can be advanced in support of the concept that acne vulgaris is in some manner related to the secretion of sex hormones, I should like to mention only the following: (1) Tars are known to cause acne lesions and to contain substances of phenanthrene structure. These phenanthrene sterols are chemically closely related not only to certain bile constituents and to ergosterol and to vitamin D, but also to the male and female sex hormones. They belong to a group of substances known to possess both estrogenic and carcinogenic properties; substances known also to be capable of producing the type of epithelial stimulation or irritation which leads to follicular hyperplasia and hyperkeratosis, and to comedones and acne. (2) There is evidence that a disturbance of iodine metabolism may play a role in acne vulgaris; for, as Rostenberg, Jr., Sher and I (*loc. cit.*) have shown, even very small doses of iodine (and bromine) regularly produce new showers of lesions in acne vulgaris; and there is certainly evidence that, concomitant with sex-gland development and during puberty, both thyroid function and iodine metabolism undergo definite alterations of adjustment. (3) Recent studies of T. Rosenthal and Kurzrock have demonstrated a low threshold of estrin in the blood and urine of females with acne. This makes it seem possible that a disturbed balance in the ratio of male and female sex hormones, and a preponderance of the male substance, may play a role in acne. This view is further supported by the fact that (as Br. Bloch has shown) acne vulgaris occurs earlier and is more severe in males; by the observation that acne often occurs in virilism; and by the observation of the physiologic hormonal changes at puberty, in which the pilosebaceous apparatus of face, back, and chest develops to a greater degree in males than in females (beard, body-hairs, etc.).

which may simulate acne vulgaris (acne rosacea, acne varioliformis, acneforme tuberculids, acneforme drug eruptions, acneforme sycosis, and acnes from external causes such as face creams and oils, other cosmetics, woolen and dyed garments, industrial oils, greases, paraffins, tars, chlorinated diphenyls, etc. In view of this, it is certain that the X-ray treatment of acne is decidedly beyond the scope of the non-specialist. And, furthermore, I repeat that in a disease like acne vulgaris, with its eventual usually good prognosis, X-ray need not and should not be used—even in the safe hands of the dermatologist—except with strictest indications, and in cases which the specialist adjudges to be resistant to other measures.

Fortunately, many mild cases of acne do not require the lengthy and often costly expedient of X-ray; and gratifying results often follow the local and other therapeutic measures which I shall now mention, measures which are all within the reach of the general practitioner and of all non-specialists.

Whatever the causal factors may be, the disease acne vulgaris, with its accompanying comedones, patulous follicles, oily or dry seborrhea and dandruff, is due primarily to a stimulus leading to a quantitative and/or qualitative change in the activity of the sebaceous glands, and to occlusion of their orifices by osteo-follicular hyperkeratosis. While X-ray is assumed to achieve its beneficial effect by diminishing the activity of the sebaceous glands, almost all local measures owe their efficacy to their faculty of removing the plugs from the duct orifices, and of preventing the formation of new plugs. In many cases of acne, I have found greases and stearic acid "creams" (ordinary cold-creams, skin and tissue foods, vanishing creams, etc.) contraindicated; for these tend to remain in the minute follicular openings, and thus to form a basis for new plugs. However, in some cases, creams containing keratolytics, such as sulphur and resorcin, are of distinct value when properly prescribed and applied.

The softening and removal of the obstructions in the plugged follicles may be accomplished in several ways.

The first way I shall mention is the employment of the softening and macerating action of hot wet dressings or of steaming the face. Almost any solution, if applied conscientiously for an hour or more daily, in the form of steaming hot towels, will achieve the desired softening of the comedones and will act as an antiphlogistic in speeding the cure of the papules and pustules. Therefore, saturated solutions of boric acid; Burow's solution 1:20; or a weak aqueous solution of alcohol (1-5 per cent) will be found effective when properly employed as hot applications. Liquor calci sulfurati (Vleminckx's solution), diluted with twenty parts water, is perhaps the most effective form of hot wet dressing. While the last-mentioned solution has the decided drawbacks of its strong disagreeable odor (H_2S), and of its action in turning the fingernails brown and of forming black sulphides on white metal reached by its fumes (silver, platinum, white gold, etc., will turn black, while nickel and chromium plate will remain unaffected), nevertheless, in severe cases, its action is so superior that I employ it regularly. The patients are, of course, to be warned of the odor; and to be told that they must put away white metal jewelry, utensils, silverware, etc.; and that they must protect their nails by wearing rubber gloves. Assiduous employment of such hot wet dressings—say one hour A.M. and P.M. for one or two weeks—will sometimes benefit cases refractory to all other measures, even including X-ray.

A second measure to achieve the desired keratolysis of the horny plugs and the clearing of the follicular orifices is the employment of keratolytic lotions and of the above-mentioned keratolytic creams. These may be prescribed alone, or to great advantage in combination with the applications of moist heat. For example, I usually order a steaming with one of the above solutions on hot towels for one-half hour before retiring, and then the overnight application of the keratolytic lotion or cream. Removal of the adherent lotion or of the cream is to be accomplished in the morning through another one-half hour of hot wet applications; or, in some cases, the morning treatment may consist of merely washing off the remaning adherent deposit of lotion or the remaining cream with soap, hot water, and a complexion brush.

In all these procedures, the desideratum is to achieve a definite but correctly adjusted keratolytic effect, one which can be maintained, but which will not prove too irritating. For this reason, it is necessary to steer a wary course between the Scylla of insufficient action, due to too weak remedies, and the Charybdis of excessive effects, due to remedies which cause unnecessarily severe irritation, redness,

XUM

soreness, and sometimes even swelling and vesiculation and severe dermatitis.

To avoid both these dangers, it is advisable to proceed slowly and cautiously. Each skin is different; and the art of the topical management of acne is to find the correct amount of treatment and the proper concentration of keratolytics to achieve the desired mild peeling. I, therefore, almost always prescribe a lotion whose strength can be delicately adjusted and varied at will, a lotion which will "turn out" as prescribed, no matter where dispensed —in preference to the almost universally employed, but often unreliable and variable, lotio alba. The following are my usual prescriptions:

(1) Resorcini 2%-10%
Sulphur precipitati 2%-10%
Zinci oxidati
Talci āā 15.0-20.0
Glycerini 15.0-20.0
Aquae
Spiritus āā ad 120.0

or (2) Resorcini 2%-6%
Sulf. Precip. 2%-10%
in Unscented Cold Cream.

One begins with the lower concentrations of resorcin and sulphur, and works up in the course of a few weeks, thus reaching concentrations which continue to effect that slight degree of dryness and almost invisible scaliness which I have learned to consider the optimum effect in most cases. The lotion prescribed can not only be adjusted in strength by varying the quantity of resorcin and sulphur, but can be made thicker or thinner, merely by varying the quantities of the zinc oxide and talcum—āā 10.0 makes a thin lotion, āā 25.0 to 30.0, a very thick one. (The latter is best dispensed in a jar).

The thicker form is desirable when patients seem to be negligent in carrying out the prescribed treatment. For the thick lotion necessitates some time for its removal, and thus guarantees the proper morning cleansing, either in the form of the morning hot wet application or the morning soap and water scrub. I have encountered not only adolescents, but also older women who, until this treatment was prescribed, had not given their face a proper washing for variable long periods reaching to many years.

In this connection it may be said that scrubbing several times a day with a rough Turkish cloth or with a soft or stiff complexion brush and with a good, bland, and not highy scented white soap and hot water is, in itself, an excellent treatment for acne vulgaris. Sometimes this alone will clear the follicles, and thus suffice to keep the dermatosis under control.

I do not know why care of the scalp should be important in managing acne vulgaris, but I have found this to be true in some cases, particularly in those with lesions near the hair margins. For this reason, the dandruff or oily scalp, which so often accompanies acne, should be treated. Shampooing not less than once weekly, and the daily application of alcoholic scalp lotions containing resorcin monoacetate (1 per cent-2 per cent) and minute amounts of bichloride of mercury (one-half per mille to one per mille) have been found effective as a scalp treatment. A useful formula is as follows:

Resorcini monoacetati	4.0
Acidi Salicylici	4.0
Hydrarg. bichlorati	0.2
Glycerini	4.0
Spiritus	
Aquae āā ad 240.0	

(In patients with blond, gray, or white hair, the resorcin compound must be omitted, as it tends to discolor and darken light hair).

Regular shampooing with a tar shampoo is, even alone, a good method to keep mild dandruff under control; and the idea that water tends to cause baldness or to "rot the hair" is no more than a superstition without basis in fact.

While acne vulgaris patients should, in general, avoid face creams, oils, greases, facial treatments, massages, etc., powder and dry rouge may be permitted in most instances. I believe that face powders containing precipitated sulphur have some beneficial action, and may tend to prevent recurrences. For young girls and women with acne, I recommend, therefore, such a sulphur-containing face powder which patients can now purchase in pleasant form and in all the various usual shades.

Unfortunately, I cannot here discuss the many ramifications of the question of the general and systemic treatment of acne vulgaris. It must suffice if I say that the patient's general condition should be brought up to par, if found low in any respect (constipation, anemia, foci of infection, etc.).

On the question of diet, I have come to hold views

which may represent some departure from the orthodox. I do not believe that fat or carbohydrate-high diets, as such, are important causes in the majority of the well-known exacerbations of acne vulgaris; but I believe that these exacerbations are due, rather, to an intolerance to specific foods which these diets may contain. Together with Fred Wise, I first expressed this opinion some years ago (Ref. Year Book of Dermatology and Syphilology, p. 10, 1933). And this view has recently received confirmation in the reports of Cleveland White and others.

The first to be mentioned among the ingested substances which I know to cause eruptions of acneform lesions, are iodides and bromides (potassium iodide, iodized salt, bromo-seltzer, etc.). Even the very small quantities of iodine in iodized salt are sufficient to cause exacerbation of acne vulgaris in many predisposed individuals and particularly in adolescents. For this reason, the indiscriminate use of iodized salt in boarding schools and colleges is a foolish and pernicious practice. As a matter of fact, in my opinion the sale of iodized salt should be regulated by law, so that a physician's prescription would be required, except in those "goitre belts" in which an increased supply of iodine may be indicated for the entire population.* While iodine and bromine are by far the most frequent offenders among the drugs, I believe that in rare instances such drugs as salicylates, barbiturates, and anilin- and pyridine derivatives have produced exacerbations in some of my patients. However, after iodides and bromides, not drugs, but chocolate (in any form) is, in my experience, surely the next most common cause of new papules and pustules in acne. Several other foods seemed to play a harmful role in certain of my individual cases (nuts, oatmeal, fish, spinach, cheese, pork, etc.).

In a very few isolated cases of long standing, which have resisted other measures, the general question of food idiosyncracy or allergy may merit more detailed and careful consideration; but, as a rule, the solution to acne vulgaris has not, in my experience, been achieved through dietary restriction.

In some cases, I have found the treatment of the secondary bacterial pyogenic factor to be useful. For this purpose, particularly in cystic cases of long standing, acne vaccines, staphylococcus vaccine, or, more particularly, staphylococcus toxoid are worthy of trial. But here, too, I must emphasize that this form of treatment is not likely to be of use in the majority of ordinary cases.

In isolated instances, other forms of treatment are sometimes helpful. Among these, arsenic by mouth (Fowler's solution) or by subcutaneous injection (sodium arsenate), injections of gold salts, of turpentine, of foreign protein, etc.—each may, in a few and obviously exceptional cases, lead to success. As before mentioned, the same may be said of treatment with anterior pituitary-like and with other hormones. In my hands, treatment with small doses of femalie sex hormone by mouth has seemed to be slightly more effective than any other endocrinologic approach. I give fifty units (0.075 mgm. of crystalline dihydroxyestrin) two to three times daily; and in some few cases the beneficial effects have been undeniable. I do not know the reason for or the mechanism of this action, but since larger doses have seemed less effective, or have even caused new crops of lesions, I am inclined to attribute the result to a desensitization (perhaps of the pilosebaceous structures), rather than to a substitution therapy.

It is undeniable that even the use of all the above measures, and even roentgen therapy, all fail ignominiously in a certain—fortunately small—percentage of cases. Because of this, and because of the expense and the time required for roentgen treatment, the search for new and simple remedies for acne must continue. A few years ago, Drs. Rostenberg, Jr., Sher and I (*loc. cit.*) suggested the use of sodium chloride (large doses, 10-15 grams by mouth, daily—in the form, for example, of the 1 gram tablets prepared by E. Lilly) in the effort to eliminate the possibly harmful excess of iodine. This treatment, beneficial in a few cases, has not proven of general value. Recently some of my collaborators and I have been experimenting with the internal administration of drugs which may be supposed to inhibit the activity of the sebaceous glands (sympatheticotropic [adrenergic] and parasympatheticotropic [cholinergic] drugs). We have also tried, for example, camphor given by mouth and by injection. Our experiments in this direction are based on the reports of camphor's effect on inhibiting the secretion of the mammary glands. (J. Rosenblatt. See also, for example, Klein, M.D., *Am. J. Obst. & Gyn.*, 31; 894, 1936).

***Fatal cases of iododermas from iodized salt have been reported. And no doubt can exist that iodized salt may suffice to elicit attacks of thyrotoxicosis and serious disease in a few peculiarly susceptible individuals.**

The sebaceous glands are, of course, close relatives of the mammary and of the apocrine glands. Our investigations in this general direction have just begun, and permit hopes rather than conclusions.

ECZEMATOUS DERMATITIS

Under the heading eczematous dermatitis, one must today include all eruptions in which other diagnoses can be excluded, and which present the essential features of vesiculation (clinical or histologic) plus one or more of the adjuvant characteristics of redness, swelling, oozing, itching, papulation and/or lichenification; and in which the principal seat of reaction can be demonstrated—by histologic study—to be within the epidermis.

In this group I here deliberately include the eczematous types of superficial ringworm of the glabrous skin, and secondary and disseminate eczematous eruptions produced by fungi and other microorganisms and/or their products. This inclusion is based not only upon theoretical considerations, but even more directly upon practical issues. For, as Wise and I have most recently set forth in our Year Book article on therapy (Modern Treatment of Common Fungous Affections, Year Book of Dermatology and Syphilology—1937), the eczematous forms of ringworm are in all probability based upon a mechanism similar to the eczematous reactions to other allergens—that is to say, the products of the fungi sensitize the skin in a fashion analogous to that in which the products of a plant, such as poison ivy or primrose, or a simple substance, such as a hair dye or formalin, sensitize the skin. For this reason, the prophylaxis of the common types of fungous eruptions most certainly does not consist exclusively in the prevention of infection—for we adults in this country are practically all infected and carry our fungi around with us, in our toe-nails and often in scarcely perceptible scaling or macerated sites between the toes, in the groin, in the perianal or intergluteal areas, etc. Logical prophylaxis, therefore, consists not mainly in employing fungicidal baths, fungicidal salves and tinctures, etc., but rather in preventing an increase in fungous allergens by arresting an increase in the amount of dead, macerated and cornified tissue and debris which may adhere to the skin. For it has been shown that fungi of the particular species in question cannot multiply in living, but only in dead tissue. It is for this reason that maceration, such as occurs when the skin is exposed to the effects of swimming-pools, showers, etc., and which often appears in intertriginous areas on the slightest friction and sweating. tends to increase the amount of culture medium available for these fungi. Thereupon, when the growth of fungi exceeds a certain limit, the fungous allergens which they produce may exceed the limit of tolerance of the given skin area, and thus produce the skin reactions which are recognized as clinical disease in the form of eczematous fungous dermatitis. The same mechanism may lead to an increased absorption of fungi and/or their products from an intertriginous focus—and thus secondarily, to the disseminated hematogenous, vesiculo-papular, and eczematous dermatophytids. I cannot go into further detail here, but must refer those interested to the summary of my views, as set forth in the mentioned article. Those who read this article will see that, in my opinion and experience, proper drying between the toes and in all intertriginous areas, the use of any mild alcoholic solution (even eau de Cologne, for example!), followed by the plentiful use of a plain absorbent talcum and kaolin powder between the toes, in socks or stockings and in shoes, is perhaps not only the simplest, but the best prophylaxis of "athlete's foot." These mild and innocuous measures will do more to prevent the accumulation of intertriginous debris, and will thus do as much to prevent outbreaks of athlete's foot as can possibly be achieved by strong fungicidal agents. For even the best fungicides now available will surely sensitize and irritate a certain percentage of users, and can nonetheless never sterilize and keep sterile the skin and nails, as far as the usual fungous flora is concerned.

Not only in prophylaxis, but also in the treatment of eczematous fungous eruptions, the production of desquamation of the skin and the removal of the fungous colonies plays an important part; so that here, too, the treatment often consists in the elimination and removal of allergens, rather than in the killing of parasites. And in achieving this desquamation and removal, the natural eczematous process itself is, in itself, often adequate; and in many other cases the natural reaction leads first to an exudative and then scaly stage which requires but little assistance. This assistance towards therapeutic desquamation can be given in the form of such mild drying lotions as calamine and zinc oxide with the

XUM

addition of 1-3 per cent resorcin; or of a very dilute tincture of iodine (one-half to 1 per cent in alcohol), or often simply with bland powders, or washing with 35 to 70 per cent alcohol; and later, in the end stages, through the application of such a mild emollient as, for example, borated vaseline.

While the above measures are, of course, more specifically intended for eczematous dermatitis in which fungi have been demonstrated as causal agents, it may here be stated that in eczematous dermatitis—whatever its cause, whether due to fungi, or to external or hematogenous exposure of the skin to so-called sensitizing agents capable of producing eczematous hypersensitivity,—the essential and broad principles of non-specific local and topical treatment remain the same. In general, it may be said that unless the cause is either obvious or easily ascertained, its discovery will require the facilities and special knowledge which are usually beyond the realm of the non-specialist; and it is therefore pleasant to be able to tell the non-dermatologist that the discovery of the etiologic agent, while undoubtedly extremely important, is not necessarily a *sine qua non* in the management of many cases of eczema.

In discussing the protean and diverse forms of "eczema" not due to fungi, I can only repeat that, whatever the cause of an eczematous eruption, the application of the proper local remedies is an essential part of the management. Here, again, superficial X-ray, while a sovereign remedy particularly in the subacute and chronic forms, should not be applied without the strictest indication or without the highly specialized technic and apparatus which are usually available only to the dermatologist.

In the topical applications, however, the general physician has a potent weapon, one which, when correctly used, will bring relief in most cases of eczematous dermatoses.

The fundamental dictum in the local treatment of all "eczema" is: the more acute, the more angry, the more highly inflamed the skin condition—the milder must be the treatment employed.

Therefore, in the acute, edematous, erythematous, vesicular, bullous or weeping eruptions, wet dressings are the first choice. However, these dressings must be correctly applied—for improper wet applications are likely to do more harm than good. There is just as much special knowledge and skill necessary for dermatologic as for surgical dressing; and dermatologic dressings are often more difficult and more tedious to apply. A wet dressing *must* be kept sopping wet. Neither absorbent cotton nor ordinary gauze is the correct substance to employ. Absorbent cotton packs down too tight and hard; and gauze is too loosely meshed, and presents too rough a surface. Old soft linen, old cotton cloth (pillow slips, sheets, napkins, tablecloths, white shirting, etc.) should be folded four or more times, soaked in the indicated solution, and applied *evenly* to the affected parts. These dressings must be literally dripping wet, and must be kept so. For this reason, they must be changed every fifteen to twenty minutes. And again for this reason, oil silk, rubber sheeting, or other impermeable material must be used to protect the bed, pillows, furniture, etc., when these dressings are being applied. In very acute conditions, the wet dressings should be kept uncovered, as the speedier the evaporation, the greater the antiphlogistic and cooling effect, and the greater and speedier the relief. But in many cases, rather than to allow drying, it may be advantageous to apply a covering of oil silk over the wet cloths, thus preventing too rapid evaporation, and thereby demanding less frequent changes of dressing.

I have gone to some length to explain what is perhaps an obvious technic. But I believe that the manner of the application of a wet dressing is even more important than the solution employed.

In many instances, the circumstances are such that it is at once obvious that no wet dressings can be used (generalized dermatoses, poor nursing facilities, ignorant or uncooperative patients, etc.). It is then preferable, in my opinion, to dispense with wet applications, rather than to let these be incorrectly used (allowed to dry, for example).

As solutions for wet applications, one may employ either Burow's solution diluted 1:20, or boric acid solution (saturated); or, if nothing else is available, milk may be used. Many other solutions are employed: physiologic saline, Thiersch solution, dilute eau d'Alibour, etc. For areas with secondary pustulation or with marked denudation and epidermal defects, I find an aqueous solution of silver nitrate ¼ per cent, ½ per cent to 1 per cent to be the unparalleled wet dressing.* Potassium per-

*In the last fourteen years I have seen these dressings used on many hundreds of patients and have encountered no case of argyria. Moreover, I know of no report of argyria from this particular use of silver nitrate.

manganate as soaks or wet dressings (1:5,000 to 1:20,000) is also useful in impetiginized, pustulating, or infected acute dermatitis (fungi, yeast, cocci, etc.).

Lotions or powdery suspensions are the next after wet dressings in mildness; and are to be used either in subacute dermatitis, or after wet dressings have done their work, or when wet dressings cannot be applied. This last eventuality is common; for many patients cannot—and more, will not—take the time or trouble to apply wet dressings. Moreover, certain parts of the body (the back or the chest, for example) cannot well be treated with compresses; so that when an acute dermatosis is generalized or affects very large areas, the wet dressing idea is best abandoned, and failing the facilities for a permanent bath, lotions or powders must be employed.

An excellent basic lotion for such purposes is:

Zinci oxidati	
Talci āā	40.0
Glycerini	30.0
Aquae ad	240.0

To this, one may add one or more of the following:

Menthol 1/8% to 1/2%
Phenol 1/4% to 1%
Acid Salicyl. 1% to 2%
Liquor carbonis detergens 5% to 20%

And in cases of subacute eczematous conditions in which fungi (trichophytons or monilias) or skin cocci may be playing a role, Cinnabar 1 per cent or resorcin 2 per cent to 4 per cent, added to the above basic lotion, often proves effective.

Here, again, the mode of application is important. These lotions should be painted evenly and freely on all the affected areas, with an ordinary two-three inch-wide flat varnish brush, obtainable in a paint or hardware store. The lotions should be applied carefully, four or more times daily.

When a generalized eruption is being treated, the above basic lotion may sometimes be modified through the use of alcohol and water, equal parts, instead of just water. This will facilitate drying. Moreover, in widespread dermatoses, the amount of menthol must be reduced, as the larger amounts often produce chills; and when resorcin or other phenolic substances are employed, the remote possibility of toxic effects must be borne in mind, and the urine be examined at regular intervals.

In subacute conditions and on sites where bandages can be applied, pastes are the next in order of mildness. A paste, due to the porosity achieved by its high content of suspended particles, has an entirely different action from that of an ointment. Pastes do not dam up the secretions to the extent that ointments do. A paste, properly dispensed and applied, is dry; and has often an effect which is drying rather than emollient, an effect more closely resembling that of a lotion than that of a grease.

Lassar's paste is an excellent standard remedy when correctly made by a capable druggist. In some cases, it may then be used even in acute and oozing conditions,—for example, in a vesiculating eczema of the hands, when wet dressings cannot be employed, or after wet applications or soaks have done their work.

Salves may be used in the subacute dry or scaly, or chronic lichenified eczematous dermatoses, when these conditions are not too widespread. It is my opinion that, for purely practical reasons, patients with very widespread dermatoses (unless they can be hospitalized) must be treated with lotions rather than with salves.

Of ointments which can be recommended, the simplest are: plain vaseline, borated vaseline, boric ointment, or zinc oxide ointment. These are not likely to be beneficial on acutely inflamed, blistering or oozing surfaces; and should not be used, as a rule, in hairy or intertriginous areas.

As the skin condition under consideration becomes less and less acute, stronger and stronger adjuvants may be incorporated in the pastes or ointments. Of the many possibilities, I can here mention only the fewest selections. These are here listed in approximate order, ranging from the most indifferent and the mildest to the stronger medicaments:

Naftalan 5% to 10%
Sulphur precip. 5% to 10%
Acid. Salicyl. 2% to 10%
Oil of Cade 5% to 10%
Crude coal tar 2% to 20%
Resorcin 5% to 10%
Chrysarobin 1/10% to 5%.

As antipruritics, one may add phenol 1/4 per cent to 1 per cent, menthol 1/8 per cent to 1/2 per cent; and, when there is no indication of a hypersensitivity, or grave fear of inducing a sensitization, 5 per cent to

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10 per cent benzocain. (In eczema, not only pre-existing sensitivity to medicaments, but also the possibility of later sensitization to previously tolerated remedies, must be borne in mind; and the local anesthetics all have a propensity for sensitizing, and must be used with awareness of this possibility.)

In very chronic, thickened, hyperkeratotic and lichenified conditions, combinations of strong remedies are sometimes useful; but these must always be employed with care and with full knowledge of their effects and by-effects.

It would, of course, be possible to go on almost indefinitely with this listing of topical remedies. I prefer to break off here, arbitrarily; and to close by emphasizing certain generalities.

The art of local treatment in eczematous dermatoses lies in the discovery of the proper remedy for the existing peculiarities of the individual case. In this, the vehicle employed (wet dressing, lotion, paste, or salve) is often fully as important as the active ingredients selected. And the correct technic of application and bandaging is the *sine qua non* of successful treatment. He who would treat eczematous dermatoses should familiarize himself with at least a few remedies. He should be well acquainted with the actual appearance, physical properties, and other qualities of his prescriptions: in other words, "how they make up". (I try never to prescribe a remedy which I have not seen, touched, smelled, and applied as a trial to my own skin.) In treating eczematous dermatitis of any kind, it is well to remember that these patients usually have their skin disease *because* they have "sensitive skins"; and that, for this same reason, they may be or may become extremely sensitive to almost any of the active remedies applied. Therefore, it is best not to prescribe any proprietary (which may contain any number of unknown sensitizing ingredients). Furthermore, it is desirable always to find out what has been previously used; and if the former remedies were properly applied and proved useless, or if they were said to have made the condition worse, these remedies and all their active constituents should be avoided in the new treatment. Begin cautiously with each new remedy; let it be applied to a small area for several days, and watch its effects. If there are several areas involved, or if the dermatosis is generalized, one may derive advantage from this situation by trying out several remedies at once. Thus, four different remedies can be prescribed at a first visit, each to be used on a different area. In this way, one quadruples the chances of finding the correct topical agent at the very first visit.*

In closing, I can only repeat once again: In eczematous dermatoses, proceed cautiously; use common sense, and know the exact qualities and effects of what you prescribe; and remember that a few simple, mild remedies, correctly used, are often better than strong or complicated agents.

I am convinced that the physician will be successful only if he himself knows precisely how each topical agent is to be used; and only if he will take time and pains to instruct the patient in the exact objectives of the treatment and in the minutest detail of the procedures of the correct technic of local application and cleansing; and if he will then carefully check the treatment, observe its effect, and, if necessary, adjust and readjust his prescriptions.

I realize that I have unfortunately no more than touched upon a few points concerning the treatment of only two dermatoses. There are, of course, many other skin conditions which the general practitioner will be called upon to diagnose and treat. Among these, I may mention seborrheic dermatitis, psoriasis, pityriasis rosea, pruritus (including pruritus vulvae and ani), furuncles, and other pyodermas. However, the ordinary dermatologic text-book gives the basis for the simple management of these conditions; and to go beyond a text-book exposition would have required more time than is at my disposition today.

I have, therefore, thought it advisable to limit myself to a more detailed discussion of the two most commonly encountered types of dermatoses; and have attempted to present certain modern aspects and certain conclusions from personal experience, which could not be found in the ordinary text-book. And even in this I have not aimed at being exhaustive, but have tried to select that which was neither too well known nor too controversial, as well as that which was not obviously the distinctive problem of the specialist in dermatology. I realize that this rigid selection has narrowed this presentation, and that I have omitted many important points. For example, I have not touched upon the differential

*For a more detailed discussion of the technic of topical therapy see the leading article in the 1935 Yearbook of Dermatology and Syphilology; and also, for example, the article by William Garbe and me, *Some Technics of External Therapy, American Journal of Nursing,* Vol. XXXVI: 9, p. 873.

diagnosis of the various forms of acne and of "eczema"; nor mentioned the details of the often all-important search for etiologies by means of history, of elimination, and of skin tests. I have also omitted many important forms of therapy, such as treatment by desensitization with various allergens. Omissions of this type have been made because I am convinced that many of these procedures are, whenever possible best left to the specialist; and because I have felt that it would be impossible here to submit information that would be sufficient to prove of practical value to the general physician.

In spite of my recognition of its possibly too narrow scope, I conclude my present discussion with the hope that at least some of the points I have tried to make will prove of actual help to general practitioners; and that some physicians will be aided in their management of the problems of acne, of eczematous ringworm, and of other "eczemas". For, if this has been accomplished, it will enable some among this audience to confer additional benefits to their patients; and will save a certain number of sufferers from seeking non-medical advice, and from receiving treatment at the hands of the ignorant and unprincipled.

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CORONARY THROMBOSIS IN A TWENTY-SEVEN-YEAR-OLD MAN.*

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Coronary Thrombosis in a person as young as twenty-seven is sufficiently unusual to warrant a report of the following case.

H.A.K., aged twenty-seven, was seen April 17, 1937, in consultation with Dr. T. E. Rucker, through whose courtesy I am allowed to report the case. He had been taken sick on the thirteenth with a presternal pain so severe that it necessitated his stopping work. Dr. Rucker saw him soon afterwards and his examination was entirely negative. He took a total of four grs. of codeine before obtaining relief—sometime after midnight. On the sixteenth and again the next day Dr. Rucker noted frequent premature beats and then felt suspicious of a coronary occlusion. There had apparently been no fever and the blood pressure was not recorded at first.

When seen on the seventeenth examination was as follows: The patient was rather stocky; the pulse was seventy-six and perfectly regular, the blood pressure was 125/80, temperature 98.2. The heart examination was negative as was the general physical examination. On account of the severity of the pain and the previous finding of premature beats, it was thought advisable to take an electrocardiogram. This showed typical curves of a recent coronary occlusion of the T-3 type. Figure one shows this tracing together with three subsequent ones which show a gradual approach to the normal type.

A detailed study of the personal and family history of the patient was made. He was twenty-seven years old, unmarried and had worked since 1928 as cost accountant in a large manufacturing concern. His father died of apoplexy at sixty-three and his mother was living, age sixty-six, and in very good health. One of his mother's sisters died at sixty-seven of diabetes and another at fifty-nine of coronary thrombosis. The patient has four sisters and one brother, all in good health.

He had been in excellent health in the past except for an attack of chorea at the age of seven, which kept him in bed for three months. Regarding his habits this patient smoked from one and one-half to two packs of cigarettes daily and drank from four to six cups of coffee. He used alcohol only over the week-end—one to two pints. He averaged eight hours' sleep, got very little exercise, was a very large eater, especially of bread, potatoes, sweets and meat. His working hours were from 8:30 a. m. to 5 p. m. with one and one-half hours off for lunch; from about the fourth to the ninth of each month his hours were considerably longer, averaging from 6 a. m. to 6 p. m. with one hour for lunch. During this period he worked under considerable pressure or nervous tension.

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*Read before the sixty-eighth annual session of the Medical Society of Virginia, at Roanoke, Va., October 12-14, 1937.

This paper and the succeeding paper by Dr. Golston are discussed conjointly.

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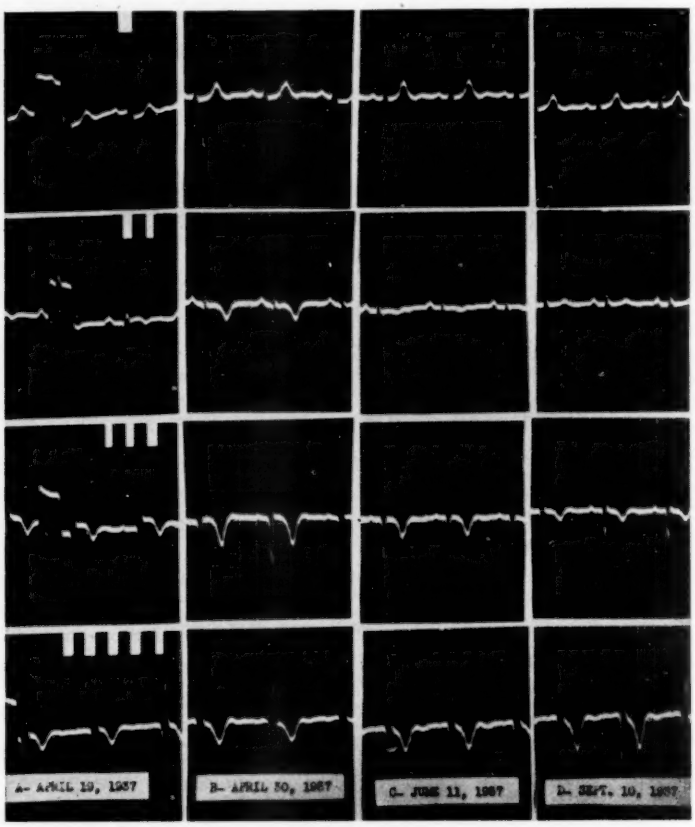
This patient was kept in bed for seven weeks. On September 17, his heart examination including a seven-foot plate was entirely negative, although his electrocardiogram was still abnormal. His B. P. was 150/90. The blood Wassermann test was negative.

Many authors in recent years have reported one or more cases of coronary disease in patients under forty. Stolkind[1] reported four cases of angina pectoris in children and twenty-five cases from the literature between the ages of six and fourteen. Levy, Bruenn and Kurtz,[2] in a review of autopsies at the Presbyterian Hospital in New York from 1910 to 1931, note the increasing incidence of coronary disease, especially in the age group twenty-five to forty-four. In a total of 2,877 autopsies, coronary arteriosclerosis of varying degree was found in 762 cases, an incidence of 26.5 per cent. Coronary thrombosis was found in seventy-two cases and nineteen of these were under forty-five. Of interest was their finding that syphilis was no more frequent in the coronary than in non-coronary cases. Durant,[3] in a series of 114 autopsied cases of coronary thrombosis, found seven cases aged thirty-five or under; all were male. Glendy, Levine and White,[4] at the 1937 meeting of the A. M. A., read a paper on "Coronary Disease in the Young". They reported a series of 100 cases of angina pectoris and coronary thrombosis in patients under forty. Of this number seventy-eight had thrombosis proven either at autopsy or by electrocardiograms. Only four cases in the whole group were females. Their paper, yet unpublished, is the most comprehensive that we have seen. Smith and Bartels[5] reported two cases of marked cardiac hypertrophy with coronary thrombosis, both male aged thirty-five and thirty-six. They present a table of twenty other reported cases of coronary thrombosis in patients aged forty or less.



Note that in D-1 the S-T segments have returned to the isoelectric line and T-2 has become upright.

In the table below we have added to theirs all

the other reported cases that we have been able to find. The age and sex are given where this is known. The predominance of the male sex is noteworthy. If we count only the reports where the sex is given, there are seventy-one males and seven females—a proportion of ten to one.† According to Smith and Bartels the youngest reported case is twelve; we have not consulted the original article. Sprague and Orgain[6] reported two cases in boys of fifteen. Jamison and Hauser's case was eighteen; May's[7] youngest case was nineteen.

It is interesting to speculate as to the cause of the increasing occurrence of coronary thrombosis in the young. Arteriosclerosis is the pathologic lesion invariably found in all of the cases referred to in this paper that have come to autopsy. Coronary embolism is an extremely rare condition, although it has been recorded. The reported cases of coronary occlusion in infants have been from this cause.[8, 9] Arteriosclerosis, however, has been found in children as young as two years (see Stolkind[1]).

Tobacco has been definitely proven to cause peripheral vascular constriction. It is a reasonable assumption, though not proven, that it may cause coronary constriction also and thus predispose to coronary thrombosis. We do know that it may cause anginal pains. Acute rheumatic fever is known to cause changes in the electrocardiogram simulating

†This is excluding Glendy, Levine and White's large series.

TABLE 1.

Authors	*Cases*	*Male Ages*	*Sex not Stated*	*Female Ages*	*Total Reported*
Krumbaar & Crowell	1	38			
Wearn	1		under 40		
Parkinson & Bedford	3		under 40		
Levine & Tranter	1	39			83
Allan	1		39		
Kerr, Larkey & Larsen	2		32, 38		15
Osler	1		35		
Klingman	1	35			
Palmer	1		36		
Nathanson	2		32, between 30 & 39		
Jamison & Hauser	1	18			
Clark	1	30			
Dreschfeld	1		12		
Benda	1		12		
Werley	1	40			
Smith & Bartels†	2	35, 36			
Levine & Brown*	3	36, 39		39	145
White	21	22, 23, 26, 26, others 31-39			418
Conner & Holt	20		2 under 35		287
Leary	10	25, 26, 28, 31, 37, 37, 38, 38, 38		34	
Barnes & Ball	2		under 40		49 (all autopsied)
Meakins & Eakin	7	6 males		1 female	62
Christian	5		31-39		71
Lisa & Ring	3		28, 30-39		100 (all autopsied)
Coelho	1	34			
Rathe	1			33	
Marie	1			38	
Boas & Donner*	7	31-40 all male			71
Riesman	1	32			88
Sprague & Orgain	5	15, 15, 29 33, 39			61 (all autopsied)
May	4	19, 38, 39		20	
Wright-Smith	10	29	31-40		495 (all autopsied)
Glendy, Levine & White	78		8 under 30		
Durant	7	23, 31, 31 33, 33, 33, 35			
White, Glendy & Gustafson	1			22	114
	208	71	130	7	

†Authors to here quoted by Smith & Bartels.
*Authors between asterisks quoted by White.

coronary thrombosis. Autopsies on some of these cases have shown coronary occlusion. However, no case of acute rheumatic fever has been included in our tabulated cases. It is extremely doubtful if a previous rheumatic fever or chorea is a predisposing factor in the early development of coronary thrombosis. Our patient was a heavy smoker, a heavy coffee drinker and a heavy eater. His work was of a kind requiring considerable nervous strain during a part of each month. In his family history there was evidence of defective tubing, as Osler called it, on each side. Glendy, Levine and White in their paper have compared the personal and family histories of their 100 cases of coronary disease under forty, with the histories of 300 persons living past 80. Their conclusions regarding the younger group are as follows: "The male sex is overwhemingly the victim. . . . A greater number of such patients may be expected to have hearts that are normal in size, than among cases of coronary disease in general. There are fewer complications and diabetes or evident peripheral vascular disease is uncommon. The duration of life for those who died and the life expectancy of the survivors is greater than in coronary disease of all ages, but the susceptibility to sudden death is just as great. Inheritance and ancestral longevity are important factors in the occurrence of early coronary disease; racial factors are no doubt also of importance in view of the high incidence of Jewish people in our series of 100 patients. Urban life, sedentary occupations and habits, possibly excess of diet, the excessive use of tobacco, over-weight and increased nervous sensitivity and strain all appear to be more predominant in cases of early coronary disease than in those who have achieved long life. Alcohol and serious infections do not seem to play an important role."

Of the above mentioned factors, it seems to me that excess tobacco and food, lack of exercise and relaxation, mental strain and especially an inherited tendency to vascular disease are more important than the others. Let us learn to live wisely and try to prevent the increasing occurrence of crippled coronary cardiac cases.

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ACUTE CORONARY THROMBOSIS.*

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Acute coronary thrombosis as we usually see it is an emergency condition which occurs at any time of the day or night and calls for a proper evaluation of the symptoms and signs in order to reduce mortality rate, which can be as high as 50 per cent and as low as 4 per cent depending upon the amount of heart involvement and reaction to the judicious use of treatment and nursing.

PATHOLOGY

Saphir[1] and his co-workers from their examination of post-mortem coronary thrombotic hearts showed that every heart had more than one occlusion or marked narrowing of the coronary arteries and that the left coronary artery was most frequently involved, with the most severe cases involving the descending branch with the occlusion taking 2 cm. from the mouth of the artery and usually at the bend of the artery.

These men found that infarction was most common in the apical region, sometimes the septum, less frequently the left posterior wall.

Occasionally there are cases of "silent attacks" in which the infarction is small, but give no clinical signs or symptoms of a pathological change.

They also demonstrate the fact that there may be an occlusion of a main branch of the coronary artery and still show no infarction of the myocardium, because of the collaterals of the coronary arteries, i.e., (1) Thebesian vessels; (2) Anastomoses between the vaso vasorum of the aorta and the coronary arteries; (3) Extracardiac coronary collateral circulation.

We mention these pathological changes in that they have a bearing on the discussions now prevalent concerning the diagnosis and treatment.

ETIOLOGY

The causative factors are the same as those seen in angina pectoris, with the exception that at least 50 per cent of cases of acute coronary thrombosis are preceded by attacks of angina pectoris and that approximately 66 per cent of cases give a history of hypertension.

Harrison[2] states that in coronary thrombosis the attack will come on, as a rule, during rest, due to the fact that the circulation is slow, thereby encouraging the formation of a clot within a vessel, while in angina pectoris the attacks come on with exertion due to a lack of oxygen to the myocardium.

CLINICAL PICTURE

Sampson and Eliaser[3] point out that a diagnosis of an impending attack of acute coronary thrombosis can be made when a patient is seen before the thrombotic attack. These patients have a continuous prolonged pain which is usually preceded by exertion, does not occur during sleep, may subside suddenly and spontaneously without the use of opiates and is affected very little, if any, by nitroglycerine. The electrocardiograph will demonstrate no changes. These attacks will precede the major attack from one to twenty-one days, average seven days, and when recognized means that these patients can be put to bed and thus lower the mortality rate to as low as 5 per cent, because, with the gradual closure of the coronary artery, an opportunity is afforded for the collateral circulation to go into effect.

During the attack of acute coronary thrombosis the patient will look shocked, facies anxious, pallor of the skin, beads of perspiration oozing from the pores, skin cold and clammy, mucous membranes ashy gray. The patient is highly excited, restless and as a rule clutches at his chest because of the agonizing substernal pain.

TYPES OF ONSET

(1) Pain.—In a series of thirty-one cases Bruenn[4] *et al* noted that 39 per cent had pain with their coronary attack. There may be a variation from slight pain to the typical acute, crushing, squeezing, agonizing sensation located around the sternum or epigastrium. This painful manifestation may radiate to the back, shoulders or arms and may last for hours or days.

(2) Respiratory.—Dyspnea may be the initial symptom and in its advanced stages orthopnea may occur. These attacks occur suddenly while sitting or reclining.

(3) Gastro-Intestinal.—We have a group, usually

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†Formerly of Roanoke, Va.

middle aged, who start out with attacks of nausea and vomiting and having a tender abdomen.

(4) Harrison[2] makes a classification of:

(a) The phenomena of hypokinetic circulation (circulatory collapse).

(b) Manifestations of dyskinetic circulation (congestive heart failure).

(5) Silent Coronary Group.—The chief complaint is generalized weakness with no typical symptomatic complex, and may be overlooked, mistreated and be the cause of death.

PHYSICAL SIGNS

(1) Fever.—This will average 100° to 102° and in those with slight fever lasts three to seven days. In those patients in a state of shock one should take rectal temperatures, as oral temperatures may be normal and rectal temperatures may be high.

(2) Leucocytosis.—I mention leucocytosis after fever because both are a result of necrosis of the heart muscle. The average count runs from 11,000 to 15,000 and may appear as early as the sixth hour from the onset of the attack.

(3) Cardiac Findings.—Usually the heart sounds are weakened and sound distant with a diminution of the first sound at the apex.

Arrhythmias may develop, auricular fibrillation being the most common, and as a rule are transient in character.

Pericardial friction rub at the apex may be heard in a small percentage of cases when the anterior coronary artery is involved, and is heard about the end of the first week and is transient in character.

Conduction defects are usually seen with posterior infarctions.

The rate may be accelerated from 100 to 120 or, as a sign of failure, there may be a tic-tac rhythm. Bradycardia is occasionally seen in organic changes of the conduction system.

Murmurs occur usually with the progression of the disease. The most common murmur is a functional apical systolic murmur as a result of left ventricular dilatation. In hypertensive cases an aortic systolic murmur may be heard as a result of aortic dilatation. Basal murmurs are infrequent.

The blood pressure usually drops in acute coronary thrombosis with a fall of both the systolic and diastolic pressures. In shock[5] there is a decreased cardiac output with the result that the systolic pressure may drop lower than the diastolic pressure and as a result a decreased pulse pressure; this is seen with pulse pressures of 20 mm. or less.

Cyanosis may persist throughout convalescence, even though all signs of heart failure have disappeared and may be evidenced as a result of:

(a) Shock—Grayish pallor.

(b) Right Ventricular Failure—Dark bluish purple cyanosis.

(c) Shock and Heart Failure—Intense blue, symmetrical acrocyanosis.

(4) Nephritic Findings.—With the excessive outpouring of fluids through the skin and the decreased blood pressure oliguria occurs. With the care of the cardiac condition the kidneys will come back and need not be a cause of great concern.

A great many will show traces of sugar and acetone in the urine, albumin and casts; this is usually the result of shock and does not require any heroic diabetic or nephritic treatment, but will disappear as the patient improves.

(5) Embolic Phenomena.—When they occur they are either the result of mural thrombi that have formed in the left ventricle, causing cerebral manifestations, or when derived from the right ventricle, pulmonary manifestations.

ELECTROCARDIOGRAPH

Saphir[1] attributes the electrical changes that take place in the electrocardiogram as being due to the damage taking place in the heart muscle with the development of ischemia to the tissues and is not a result of the fibrous replacement of heart muscle tissue.

The serial[6] changes that take place in the standard three leads are seen in 90 per cent of patients. When this routine shows normal Feinstein[7] and Lieberson show that in lead four, when serially taken, will give a definite clue as to the diagnosis. These electrical changes may appear on the electrocardiogram within an hour or two with infarction or appear days later, usually between the fifth and tenth day. The coronary T-wave may resume its normal appearance within six weeks to twenty months after an attack occurs.

Barnes[8] and Whitten classified the location of the infarcted area by the changes in the electrocardiogram; however, Gilchrist and Richie disagreed because of the results obtained in their post-mortem findings. A classification can be made as follows:

(a) Anterior infarction—involving the apex.

Lead 1—elevated S-T wave, with subsequent

inversion and coving of the T-wave and appearance of Q.

Lead 3—reciprocal change with depressed S-T wave.

Lead 4—absent Q-wave, upright coved T-wave.

(b) Posterior infarction—involving the base.

Lead 3—elevated S-T wave with subsequent inversion and coving of the T-wave and appearance[9] of Q.

Lead 4—large[7] Q-wave and T-wave is unusually deeply inverted.

(c) Lead 4—after the acute stages are over, Lead 4 may give information for months and years later as to previous coronary attacks, absence of Q-wave and upright T-wave is positive evidence of previous anterior infarction.

(d) Low ventricular complexes in some acute coronary cases.

(e) Large P-waves may be seen as a result of the load placed on the auricles in heart failure.

MEASUREMENTS OF CIRCULATION

Robb[10] and Weiss describe the interrelation and significance of the circulatory tests, i.e., in failure of the left ventricle; the first sign is a diminished vital capacity, then a slowing of the blood flow and pulmonary congestion; this results in a prolonged arm-to-tongue circulation. With the involvement of the right ventricle, the venous pressure becomes elevated and finally the peripheral flow is slowed and the arm-to-lung time is increased. These tests are:

(1) Vital Capacity of Lungs—is an accurate measure particularly when it is below 60 per cent of normal.

(2) Arm to Tongue Time—

(a) Left ventricle function, using saccharine intravenously. The normal time averages twelve seconds; when eighteen seconds or more, it is indicative of slowing of circulation.

(b) Right ventricle function, using ether intravenously. Normal time averages six seconds; this is prolonged with slowing of venous return to the right ventricle.

(3) Venous Pressure—indication primarily of failure of the right ventricle, evidenced by distended neck veins. Venous manometer used (direct method). Pressure of nine cm. or higher abnormal.

These simple tests are cited as an accessory aid in the prognosis and treatment.

At this juncture I wish to give a short résumé of three cases, all of the Jewish race, males, and average age fifty-one.

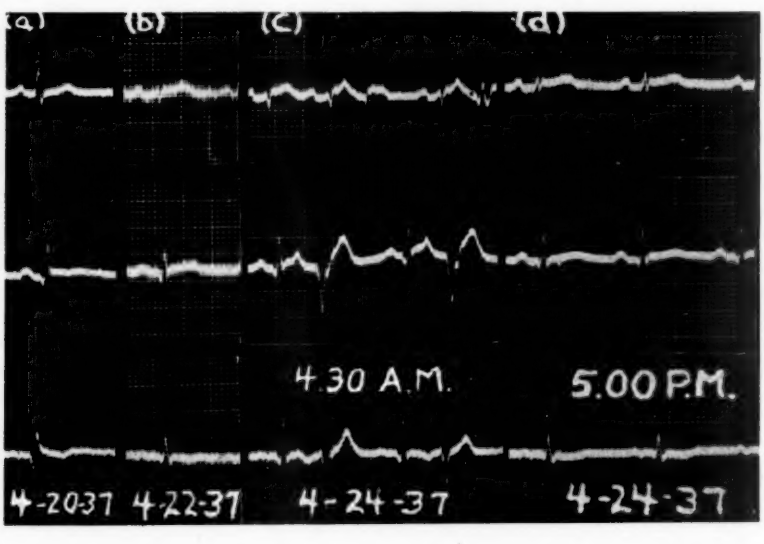
Case 1.—C.H.L.—Age forty-five. This patient had his attack July 20, 1933. While driving he had a sudden onset of agonizing pain in the mediastinal area. Pain traveled down both arms; he was dyspneic and cyanotic; pulse sixty; blood pressure 190/90. Was nauseated and vomited, temperature of 101; W.B.C. ran from 10,000 to 18,000 during the first week; he had an eosinophilia of 2 per cent. The cardiac sounds were diminished in intensity. This is a typical case which required a great deal of morphine to relieve pain. The first week, however, with the substitution of aminophyllin the pain was relieved and no narcotics were required. The patient remained in bed six weeks before he was allowed in a chair and when discharged the blood pressure stayed at 120/80. This patient is a leather goods salesman and is pursuing his normal occupation without any evident signs of a previous attack of coronary thrombosis. An electrocardiogram taken December 16, 1935, almost two and one-half years after his attack, shows a significant persistence of an increased take off of the S-T wave above the isoelectric line in Lead two. The low T-waves of Lead one and the high take off of S-T wave in Lead two are indicative of coronary disease.

Case 2.—M.B.—Age fifty-three. Seen April 10, 1935, and had an attack similar to Case one. Occurred while asleep. Had a history of hypertension 230/?; when seen blood pressure was 120/70. W.B.C. 15,280 and temperature 100.4° on the second day. He had a 3 per cent eosinophilia. This patient acted like a patient with gall-bladder colic; he was orthopneic, and deeply cyanosed. Massive doses of morphine to the point of toxicity had to be given to control his pain. His dyspnea and cyanosis was relieved by an oxygen tent; however, it did not relieve his pain. 20 per cent glucose (150 cc.) intravenously was given because of its caloric value. This patient improved with good nursing care while at the hospital, but through the insistence of the family and against my judgment was allowed to go home two weeks later. This patient was well on the road to recovery, but, due to ignorance and lack of cooperation, during the third week patient attempted to walk up a flight of stairs and collapsed, returning to the hospital the next day showing signs of cardiac decompensation with murmurs, systolic in time heard at the valve areas, and on the third day

a pericardial friction rub was noted. I could not get the family or patient to cooperate with me in my endeavor for enforced rest and quiet and requested that they use another doctor, which was done. Subsequently I heard that the patient finally developed into a cardio-renal case and died five months later.

Case 3.—H.T.—Age 53. Seen April 20, 1937, two days after his attack, in consultation with Dr. C. Long, in Johnson City, Tennessee. This patient had the typical coronary attack and attendant substernal pain, pain down left arm, was ashy gray, and had dyspnea and shock. The patient acted like an angina pectoris condition; however, he had a temperature 101.2° and a leucocytosis of 11,350. The heart sounds were diminished in intensity, and his blood pressure had dropped from 170/? to 90/60. A transient pericardial friction rub was present on the fifth day. This patient was treated with massive doses of morphine and oxygen, and on April 24, 1937, was given digitalis and adrenalin when he was in a state of complete circulatory collapse. The patient was kept on digitalis for two weeks and kept in bed for three months. My last information is that the patient is walking about.

The electrocardiograms taken on this patient show the S-T changes with the passing of the days:



(a) April 20, 1937—Two days after the attack, there is slight elevation of the S-T wave above the base line in Lead two.

(b) April 22, 1937—The S-T wave becomes higher in Leads one and two with a lowered voltage of the complexes.

(c) April 24, 1937—4:30 A. M.—Patient in circulatory collapse. Electrocardiogram taken immediately after digitalis and adrenalin had been given hypodermically showed S-T take-off at its highest stage and definite coving of the T-wave in Lead one with ventricular extrasystoles in Leads two and three.

(d) Same day as (c) preceding, another electrocardiogram was taken at 5:00 P. M. The patient was on digitalis gr. iss. t.i.d. The patient shows a splendid response to digitalis in that the ventricular extrasystoles have disappeared, the rate and volume being regular and slower. The S-T wave elevation was evident in Leads one and two and depressed in Lead three. We could classify this as an anterior infarction.

In an analysis of these three cases one notes the evident similarity in the signs and symptoms and that a diagnosis can be made with a certainty.

PROGNOSIS

One has to be guarded in their prognosis because of the high mortality rate which is influenced by the following factors:

(1) In the first attack without heart failure Masters[5, 11] *et al*, with a group of 140 cases, had gotten a mortality rate of 4 per cent, the mortality

rate jumped to 30 per cent with heart failure. With multiple attacks the mortality rate increased to as high as 65 per cent.

(2) Levine states that with a persistence of congestive heart failure through a period of weeks, the condition assumes a grave prognosis.

(3) Wood[12] and Wolferth mention that a posterior infarction has a better prognosis than anterior infarction and subsequent recovery is apt to be more rapid and more complete.

(4) Masters[5] and his co-workers, in a study of their results, note the following:

(a) Prognosis is better with a slow pulse rate, and mortality is higher with a heart rate of 120 or more.

(b) With faint or poor heart sounds, failure was found in 4/5 of patients and mortality rate was 29 per cent.

(c) Those developing gallop rhythm had a grave prognosis, this being observed by Thompson[13] and Levine.

(d) When the respiratory rate reached twenty-eight and above, the vital capacity was diminished and the incidence of fatal attacks rose.

(5) The blood picture will show the following:

(a) According to Masters, the prognosis is poor with a persistent leucocytosis and a 20,000 or 10,000 count does not alter the prognosis.

Contrary to this, White feels that the grade and duration of the leucocytosis is indicative of the size of the infarct and the prognosis.

Goodrich[14] and Smith stated that (1) a rather high leucocyte count after occlusion was of serious significance. (2) A non-filament percentage curve above 30 per cent after the fourth day was indicative of a large area of infarction and poor prognosis. (3) A lack of eosinophiles or an eosinophile curve not rising above 1.5 per cent in the first ten days was of unfavorable import. (4) A non-filament percentage below 25 per cent and an eosinophile percentage rising above 3 per cent was suggestive of a less extensive infarct and favorable prognosis.

(b) Rabinowitz[15] *et al* show that the sedimentation rate is most marked on the third to fifth day and will decrease with the improvement of the patient.

(c) Steinberg noted that with a persistent non-protein nitrogen or rise the prognosis was serious.

(6) The average[16] duration of life is two years but those who have an uneventful recovery may live a normal life for many years. Therefore, the prognosis must be guarded and yet hopeful.

TREATMENT

In the treatment of this condition we must bear in mind that there is a variable change in the physical condition of these patients and at times it is extremely difficult to appraise the value of drugs used. With this in mind, we note these salient features:

(1) Rest.—It is agreed by all that absolute rest is imperative even though the diagnosis is questionable. As seen in Case two, the patient who leaves bed too early may develop congestive failure or may have a recurrence of an attack.

(2) Morphine is the most important drug. It should be given hypodermically for the relief of pain and the effect of lowering metabolic activity. Morphine should be given in large doses—to the point of toxicity—giving ¼ grain and if necessary repeating it in fifteen minutes to a half an hour, the dosage being dependant on pain and rate of respiration. Levine states that this is one condition that you need not be fearful of habit formation as these patients do not crave the drug following convalescence. Masters[5] favors the use of dilaudid, claiming it is less constipating than morphine itself. Morphine should not be given with mercurial diuretics as it has an antagonistic action.

(3) Digitalis.—It is commonly agreed that the use of digitalis should be avoided as it may precipitate ventricular tachycardia, fibrillation or ventricular rupture. On the other hand, it is given in (a) circulatory collapse, as in Case three; (b) persistent auricular fibrillation, and (c) congestive failure.

(4) Quinidine.—Masters[5] is against the use of quinidine because it is a protoplasmic poison and will cause harm to heart muscle even though it may stop arrhythmia or ventricular tachycardia. Contrary to this viewpoint, Kilgore[17] prescribes quinidine gr. III t.i.d. as routine for two months to prevent ventricular and auricular fibrillation. Levine feels safe in giving the drug as a routine the first week as he feels that death if it occurs will be due to ventricular complexes. Contraindication to the drug he believes should be evidences of conduction block, i.e., heart block and ventricular block.

(5) Xanthine derivatives.—There are conflicting opinions concerning the use of the xanthine derivatives and the various vasodilators.

Wiggers[18] and Green in their experimental work come to the conclusion that vasodilating drugs are of no benefit after complete coronary occlusion. Fow-

XUM

ler[19] and his co-workers in their experimental work find value in the use of the xanthine derivatives.

Smith[20] *et al* feel that the good and bad results from the use of xanthine derivatives, i.e., theophyllin and theophyllin ethylenediamin are dependent upon whether the collateral circulation functions. Smith believes that the best results are obtained if used in the early stages of the occlusion in conjunction with other drugs. We might say that we feel aminophyllin was useful in Case one where the patient stated he had gotten relief of his pain, and in Case three there was no apparent value to the use of the drug.

(6) Oxygen therapy has its definite use in air hunger of dyspnea, cyanosis and pulmonary congestion. In some cases it may relieve pain.

(7) Glucose.—Marvin of Yale advocates the use of 50 cc. of 50 per cent solution in order to maintain the glucose content of muscle and to prevent nausea and vomiting. Levine believes that he gave pulmonary infarction to a patient as a result of a thrombosed vein due to glucose.

(8) Saline solution, 1000 cc. subcutaneously, may be given the first twenty-four hours to replace the great loss of fluids and salt through perspiration. It has its use also in cases of nausea, vomiting and oliguria.

(9) Caffein sodium benzoate may be given with those suffering with respiratory embarrassment, such as air hunger and dyspnea. It is best to give it in the hip intramuscularly rather than the arm as there will be less soreness to the patient.

(10) Adrenalin has its use in syncope, heart block or circulatory collapse. The drug is dangerous to use otherwise.

(11) Venesection is occasionally done in pulmonary edema, increased venous pressure or hypertension.

(12) Diet.—These patients should be placed on a low caloric diet, the purpose being to lower metabolic activity, decrease cardiac output, and decrease the pulse rate. Masters[11] and his co-workers have the following routine: (a) very little food should be given during the first few days; (b) fluids should be limited from 1000 to 1200 cc., providing they are not perspiring freely; (c) those having nausea and vomiting are given no food, cracked ice or charged water; (d) with improvement over a period of three to six weeks a diet of 750 to 850 calories balanced as follows: carbohydrate 100, protein 50, fat 20; (e) adequate vitamins and calcium.

(13) Bowels.—During the first two or three days it is best to leave the patient alone, the attendant fuss and strain being not good for them; after they have quieted down one can give an enema and a mild cathartic.

DON'TS IN TREATMENT

(1) Don't treat a syphilitic during these days; leave them alone. Samuel A. Levine insists that antiluetic treatment should not begin until after the critical stage.

(2) Insulin should be avoided in diabetes as the work of the heart is increased 20 per cent when the blood sugar drops too low. They are treated by starvation, and insulin is only used in the critical cases with acidoses.

(3) Intravenous cholecystogram should not be done in coronary sclerosis or angina pectoris because an attack of coronary thrombosis can be precipitated. The dye should be given orally.

(4) No bathroom privileges should be allowed until well on the way to recovery.

SUMMARY

(1) The majority of patients survive their first attack by judicious treatment, i.e., rest, and the proper use of drugs.

(2) Those patients giving a previous history of hypertension and having a fall of blood pressure during the attack, will occasionally maintain the appearance of a normal pressure after the attack.

(3) A patient may have angina pectoris, then develop coronary thrombosis, recover, and have no further angina because of the removal of the offending vessel. Another group will get anginal attacks after recovery—to reappear on exertion.

(4) The greatest mortality is in the group that have multiple attacks and develop cardiac failure. These cases become the difficult ones and tax the ingenuity and medical skill of the attending physician.

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DISCUSSION†

†Discussion of the conjoint papers by Drs. Scott and Golston.

DR. B. P. SEWARD, Roanoke: Coronary thrombosis is a disease we formerly regarded as occurring in people above fifty years of age. During the last two or three years we have learned from a considerable number of reports in the literature that coronary thrombosis may occur in people under forty years of age. The refinements in diagnosis have enabled physicians to make the diagnosis in young people in many instances in which it otherwise would not be made.

Dr. Scott's paper is interesting because it shows careful study of his patient and also because it is among the first if not the first report in Virginia of coronary thrombosis occurring in a young person. What may be the chief cause or causes of the apparently increasing incidence of coronary thrombosis in the age group from twenty-five to forty-five years of age, to which Dr. Scott referred, is largely speculative. A familial tendency appears to be well established; in fact, it appears to be the most important etiological factor, in the light of our present knowledge. The individual's habits and sedentary life seem to be a causal factor. Perhaps, also, as Levine suggested, there may be in some instances an unusual configuration of a coronary artery or of one of its branches, causing a section of that artery to be subjected to greater strain, with the result that it early undergoes degeneration. As the degenerative process increases, the walls of the artery become thicker, its lumen more narrow, and sooner or later a thrombus forms.

The life expectancy of young people who have had coronary thrombosis is an interesting question. According to Glendy, Levine, and White, the "duration of life for those who died and life expectancy in those who survived is greater than for coronary disease of all ages". They also stated in reporting a case one month ago that "recovery even after extensive infarcts is the rule in youth". If this is true, the outlook following an attack of coronary thrombosis in young people is more favorable than we formerly thought, for it was the consensus of opinion until very recently that the younger the patient the less is his chance for recovery. Such an opinion seems to have been based largely on the supposition that the collateral circulation in the myocardium improves with the passing of the years and the older the patient the better are the chances for the re-establishment of circulation in and around the area of infarction.

Whether we can do much to prevent the increasing incidence of coronary disease remains to be seen. I am rather skeptical about it. We may try to live prudently, but with so many demands made upon us, causing life to be more and more complicated and the environment often inharmonious, the question arises, how can we avoid much, if any, mental and physical strain? The increasing incidence of coronary disease is a high price we are paying for the advancement of civilization.

DR. E. G. SCOTT, Lynchburg: Dr. Golston has given us an excellent summary of our present knowledge concerning coronary thrombosis. There are just two or three points on which I wish to comment.

He spoke of the different types of onset of an attack We should keep in mind that approximately half of the cases have no initial pain, the presenting symptom being sudden dyspnea, cardiac asthma or weakness. The three most important things for diagnosis in a suspected case are: A fall in blood pressure, the development of fever, and leucocytosis. No single sign is pathognomonic unless it be the electrocardiogram.

Dr. Golston stated that approximately 66 per cent of cases give a history of preceding hypertension. It is my impression that this figure is a little high. In Conner and Holt's series of 287 cases a previous hypertension was

XUM

noted in only 34 per cent. Apparently a normal blood pressure is no guarantee that there may not be arteriosclerosis of the coronary arteries.

It is interesting to observe the gradual rise in blood pressure following an attack of coronary thrombosis in patients with a previous hypertension. I have had a number of patients whom I have followed for months after an attack. Some of these have had repeated thrombotic attacks when the blood pressure had reached a mark close to its former level. I think the prognosis is worse in these cases than in those where the blood pressure remains at a lower level.

We have been warned not to use digitalis in coronary occlusion unless subsequent congestive failure develops. Cases have been reported in which anginal attacks developed during digitalis administration and ceased when the digitalis was stopped. Of course, during the acute phase of a coronary occlusion the objection to digitalis is that it may stimulate the heart and place additional strain on the weakened myocardium. However, I do not believe we use digitalis enough in our coronary cases after the acute phase is over. Several of my patients have had a persistent tachycardia and generalized weakness following the attack and have been remarkedly benefited by taking digitalis.

No discussion of treatment is complete unless mention is made of Dr. Beck's operative efforts to improve the coronary circulation. This is still in the experimental stage but offers some promise of benefit to the chronic coronary cripple.

I am much more interested in the prevention than in the treatment of coronary thrombosis. Let us hope that future study will show us some definite means of preventing this terrifying accident that comes so often like a thief in the night when least expected.

DR. PAUL D. CAMP, Richmond: I enjoyed Dr. Scott's and Dr. Golston's papers. I should like to ask Dr. Scott if there was any history of previous hypertension in the case he reported in which the blood pressure went up afterwards. I presume that there was some hypertension previously.

As to the question of tobacco in angina or coronary occlusion, I do not think it has been proven very definitely at all that tobacco has anything to do with it. I personally do not smoke, so I am not prejudiced about it one way or the other, but I have never been able to find any real evidence that tobacco-smoking has anything to do with causing it. Some of these cases were Jewish people, and perhaps if they had a thrombo-angiitis obliterans (a mild state, possibly) the same process took place in the coronary arteries.

As to alcohol, as Dr. Golston said, it has not been proven that it has anything to do with producing coronary thrombosis and it might possibly have some effect in preventing it.

There is one fact that I do not believe was mentioned in connection with the increased incidence of coronary occlusion. I think the fact that more people are living to the age when they get coronary occlusion may probably have something to do with the increase in this disease.

I did not quite get Dr. Golston's statement about Lead 4 and what percentage of cases Lead 4 will pick up that are normal in the three conventional leads. I guess by "circulatory collapse" Dr. Golston meant general collapse with heart failure. I do not believe digitalis does any good whatever in circulatory collapse but may do harm, although I think it does do good in congestive failure. As to xanthine derivatives, I believe Dr. Harry Gold reported that in myocardial infarctions produced experimentally in cats aminophyllin had no effect on the size of the infarct.

DR. DOUGLAS G. CHAPMAN, Richmond: There are certain pathological findings which are similar in a few cases of thrombo-angiitis obliterans, malignant hypertension, and coronary occlusion. Tobacco has been well established as a factor, and is believed by many to be an etiological one, in Buerger's disease. I believe there is much to be learned about the toxic symptoms and the pathology produced in the arterial system by tobacco. Ergotamine tartrate found in varying quantities in wheat, rye, corn and cereals may likewise prove to have an etiological role like tobacco in sensitive individuals, produring vascular spasm and arterial pathology. McGrath's experiments have proved that female rats are protected, possibly by the female hormones, from many of the arterial spastic phenomena. Tobacco smoking produces vasoconstriction of the vessels of the hands and feet, with a drop in temperature. In many cases it produces an increase in the pulse rate and blood pressure, slowing of the circulation, palpitation, breathlessness on slight exertion, and precordial or substernal pain. There may be tingling pain in the feet, and other signs and symptoms of anoxemia. If these experiments and clinical observations are correct, why should the vessels of the heart be excluded from a similar response?

DR. SCOTT, closing the discussion on his part: In reply to Dr. Camp's question, I do not know anything about the blood pressure of this patient before the attack. I believe, however, that it was elevated, because since the attack, it has risen gradually to 150/90.

Dr. Chapman has spoken of tobacco and told us the reasons why we believe it has some bearing. Dr. White advises all his patients to discontinue smoking. I know if I had an attack I would stop it. How do we know that some cases are not due to thrombo-angiitis obliterans, anyway? I should certainly advise patients to leave off smoking.

As to the statistics I quoted, they show very definitely that there is an increase in the lower age group—twenty-five to forty. This throws considerable doubt on the statement that the increase is due to people's living longer, because the increase has been found in the younger group.

DR. GOLSTON, closing the discussion on his part: I wish to thank Dr. Scott, Dr. Chapman and Dr. Camp for discussing my paper.

Dr. Scott mentioned that 66 per cent is a high percentage of cases having previous hypertension in acute coronary thrombosis. Those are not my own figures, and are quoted

from Saphir. I want to state, however, that in my limited group of acute coronary occlusion all these patients had hypertension, and with the attack the blood pressure dropped, and two out of three have maintained a normal blood pressure up to the present date.

The question of hypertension after the attack brings to my mind the thought Dr. Scott mentioned that this young individual had hypertension after the attack. There was a question in my mind if the rise in pressure was not a part of the pathological changes that attend a heart undergoing hypertrophy.

As to the question of tobacco I agree with Dr. Chapman. Harkavy has done quite a bit of work along this line and in his experience there is a distinct vascular effect. Clendening cites the case of a patient who was told not to smoke and was getting along very well. One day he felt particular good, smoked a cigar, and fifteen minutes later dropped dead.

You can get a diagnosis in 90 per cent with the usual three leads. The other 10 per cent, which are questionable, can be checked up with Lead 4. Of course, however, you cannot make the statement that by using the standard three leads and checking up with Lead 4 you will get a diagnosis in every case of coronary thrombosis.

I again want to thank the gentlemen.

TREATMENT OF BURNS IN THE HOME.

MALCOLM H. HARRIS, M.D.,
West Point, Virginia

There is no subject of more general interest and application than the treatment of burns. It is the domestic accident of great frequency, and will be confronted by the general practitioner in the course of his every day calls, far too many times, for it is the one surgical condition that almost without fail falls into the hand of the general practitioner and is seen by him first—at home or at the place of occurrence. Our personal interest in the subject has been stimulated by the number of cases seen in connection with industrial work during the past fifteen years, and the very satisfactory progress made in both shortening the period of disability and in lowering the mortality rate with the tannic acid therapy as compared with the other treatments as hitherto used by the general practitioner.

HISTORICAL

In 1815, Baron Boyer, in his work on surgery, wrote that there was no disease whose treatments accord less with theory. They were long treated empirically, and each practitioner boasted of success of particular remedies—one emollients, another astringents and repellents, but the curative indications of burns are to be drawn from the different degrees of the disease. His prime remedy consisted in soaks or compresses saturated with Goulard's water, which was it appears a solution of lead acetate in water. This evaluation of the empirical treatment could well have been written a century later, for no disease with the exception of poison oak ever enjoyed so many inefficacious remedies.

In 1925, Davidson reported the use of an aqueous solution of tannic acid, as a spray or with compresses. Since then many other solutions have been proposed and tried with the idea of coagulating the damaged underlying tissues and fixing the toxic substances *in situ*, hence preventing absorption. Combinations of antiseptic chemicals and fixing solutions have been used with more or less degree of success.

CLASSIFICATION

For our purpose, a classification of burns with reference to the causative agent would seem rational. Any agent capable of producing heat will cause a burn, and readily falls under the head of physical, chemical and electrical, each with the three surgical degrees of severity.

1. Physical
   - a. Light ray, sunburn.
   - b. Mechanical, friction burns, chafing, etc.
   - c. Fire or any direct heat.
   - d. Scalding with hot water or steam.
2. Chemical
   - a. Acids, commercial acids, sulfuric, nitric, etc.
   - b. Alkalis, lye, caustic soda or potash, etc.
3. Electrical
   - a. Electric flash.
   - b. Cauterization, from contact with high voltage.
   - c. X-ray.

FIRST AID TREATMENT

The excruciating pain of the most trivial burn would prompt one to turn first to alleviating the intense suffering. Nothing soothes so quickly as morphine, which will help prevent shock. Keep the

areas of the burns free from contamination; they are clean, and infection arises from mistreatment. Until something can be done, cover them with sterile gauze. It is seldom the case, however, that the doctor arrives in time to prevent the goodly offices of a neighbor from applying her favorite grease or powder, sometimes both, leaving a potentially infected wound. Keep the patient well wrapped and avoid chilling; the complications of burns are more to be dreaded than the burn.

GENERAL TREATMENT

The general treatment consists in local care and systemic measures. It may be assumed that the patient is coated with grime and dirt, or the first aid has consisted of an ointment applied over the burn, all of which will have to be removed before other measures are instituted. Green soap as a general agent will prove worth while in getting off the dirt. All grease must be removed in order to apply the tannic acid successfully. Chemical burns should be washed with an antidote in weak solution or freely with running water, until all the chemical is dissipated.

Tannic acid in aqueous solutions varying from 2 to 10 per cent have been advocated. For sometime a 25 per cent solution in water has proved satisfactory, with exceptionally good results. This is applied with a spray gun at hourly intervals until the part is well tanned, and then every few hours until a coat of tannin is encrusted over the burn. When the new tissue is covered with skin the crust may be removed little by little. The deeper burned areas will probably have to be treated as small ulcers. The matter of adhesions, contractures and disabling scars will have to be constantly borne in mind and every bit of one's mechanical skill and ingenuity will likely be used in preventing their occurrence.

As stated before, the complications of burns are more serious than the burns, and every effort turned to preventing their occurrence deservedly should have place in the systemic treatment. The measures to lessen pain, secure rest, and some comfort for the patient tend to thwart the dangers of shock. The maintenance of adequate body elimination by inducing the patient to take large quantities of fluid assist in the restoration of body functions. The kidneys should be carefully watched for blood and other signs of resulting derangement.

The common complications which give much trouble are pneumonia, toxemia, cellulitis, etc. No wary physician lets himself get caught with a complication if circumstances permit its avoidance.

The use and application of the coagulating therapy in burns has lowered the death rate. The old theory of mortal burns has been changed and the amount of body surface plays much less important part in the prognosis of every case.

CONCLUSIONS

1. Burns may be treated successfully in the home and the use of tannic acid therapy in some of its modifications will prove most efficacious in treatment.

2. The complications of burns are largely preventable and should constantly be borne in mind from the first, and treatment to that end should be employed.

3. The use of tannic acid therapy has materially lowered the death rate and reduced the period of disability in burn cases.

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HYPODERMIC USE OF ADRENALIN FOR DIFFERENTIAL DIAGNOSIS BETWEEN ACUTE PYLOROSPASM AND RUPTURED PEPTIC ULCER.*

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It is the desire of the writer to differentiate between acute pylorospasm and ruptured peptic ulcer by a therapeutic measure so simple that any physician can employ it to prevent unnecessary emergency laparotomy on his patients for ruptured ulcer when this condition does not exist.

Last year at Tarboro, N. C., we had the pleasure of hearing Dr. Charles W. Mayo talk upon gall-bladder disease and gallstone colic. In his talk Dr. Mayo stressed the fact that gallstone colic might be relieved by the use of nitrites. J. W. Hundley,[1] of Philadelphia, Pa., claims experiment has shown that this group of drugs exerts a dilating and relaxing action on the musculature of the bronchial tubes, and possibly a similar action on the muscle fibers of the bile ducts and the ureters directly and not by way of the autonomic nervous system.

In contrast to the direct action of the nitrites, adrenalin relaxes unstriped muscle fibers by stimulating the sympathetic nerves, the dilating action of which opposes the constricting action of the vagi. These two subdivisions of the autonomic nervous system in health maintain normal tonus in the circular passages such as the bile ducts, bronchial tubes, stomach, pylorus, and intestines. When in disease this normal balance is lost and the vagus gains ascendency, spasm may occur in the muscles supplied by the vagus, and this spasm may be local or extensive in area.

Over-activity on the part of the vagus motor fibers may occur as a result of inflammatory stimuli which are picked up by its sensory fibers, transmitted to the vagal centers and relayed back down the motor fibers to produce spasm in the parts supplied by these same vagal motor fibers, as exemplified in gallstone colic, bronchospasm and pylorospasm. Endocrine disturbance may also produce imbalance in the autonomic nervous system.

Adrenalin is a normal stimulus to the sympathetic, and adrenalin by hypodermic injection stimulates the sympathetic fibers to overcome and neutralize the dominant constricting action of the vagus and thereby to relieve spasm caused by an overactive vagus. This therapeutic action of adrenalin is employed for the relief of bronchospasm and can be used in other fields with valuable and sometimes spectacular results.

In *Annals of Surgery*, 1928, Page 669 was published an article by Dr. Charles H. Mayo,[2] entitled "Division of the Vagi for Pylorospasm," in which appeared the statement that some association, reflex in nature, exists between chronic appendicitis, diseases of the gall-bladder, spasm of the pylorus and peptic ulcers. Just what was the physiology involved was not known. It may be concluded that Dr. Mayo believed that spasm in the muscles of the stomach, pylorus and duodeum contributes to the formation of ulcer, because of his question, "may not the pyloric spasm precede the ulcer?"

To correct this condition Dr. Mayo described an operation devised by Payr, of Leipsig, which has been performed in this country by himself and by the late Dr. John B. Deaver. This operation divided the filaments of the vagus proximal to and providing nerve supply to the pylorus, thereby intercepting vagal nerve impulses which were producing pylorospasm.

In my paper, "Ileocecal Delay and Vagus Reflex as Etiologic Factors in Bronchial Asthma,"[3] read before the District of Columbia Medical Society in November, 1920, and later before the Southern Medical Association in November, 1922, and, in my paper entitled "Etiology, Mechanism, and Treatment of Asthma",[4] published in *American Medicine*, October, 1934, I described fully the reflex action through the vagus, caused by stimulation of the vagal fibers, which stimulation was incited by diseased conditions in the appendix, gall-bladder and other regions and resulted in reflex spasm of the bronchial tubes. Analogy was drawn between the nervous mechanism involved in bronchospasm, gallstone colic, and pylorospasm. Instead of at-

*Read before the forty-second annual meeting of the Seaboard Medical Association of Virginia and North Carolina at Virginia Beach, Va., December 7-9, 1937.

tempting to intercept irritating stimuli by severing vagus fibers, it was the writer's practice to correct or relieve the original source of the irritation, that is, remove the offending appendix or gall-bladder, correct conditions in their respective areas and also to treat all other portals offending the vagi.

Rivers, of Rochester, Minn., wrote as follows:[5]

"It is therefore conceivable that influences of neurogenic origin which seem capable of influencing periodic reactivation of ulcer, and which can prevent the healing of such lesions, and which at times produce an ulcer-like syndrome, may be of some importance in establishing a local chemical and spasmophilic gastroduodenal condition favorable to the development of ulcer. Somewhere within the mechanism which influences the sympathetic and para-sympathetic control of the upper part of the gastro-intestinal tract is a wealth of information which will need to be thoroughly understood before the final analysis of the condition of ulcer will be possible."

Gilson Colby Engel[6] contributed the following statement:

"Reflex Pylorospasm.—This condition is represented by a group of cases with reflex spasm from the gall-bladder, appendix, kidney, etc. These cases deserve a thorough study to find the causative factor and then the removal of the cause. This treatment in itself usually will clear up the spastic pain without further care."

In this statement Engel suggested a solution of Rivers' expressed hope, which solution I had offered in 1920, in 1922,[3] and, again in 1934,[4] to relieve spasm resulting from vagal over-activity.

With full appreciation on my part of the action of adrenalin and of the neurophysiology involved in the mechanism of bronchospasm and pylorospasm, Dr. Charles W. Mayo's talk at Tarboro on the action of nitrites prompted me to report in this paper the hypodermic use of adrenalin employed for acute pylorspasm and to suggest it for gallstone colic. It has not been my good fortune to see a case of gallstone colic recently but I have had occasion to treat, within the past two years, five cases of very acute pylorospasm. You will not be bored by long case histories for the symptoms were very similar. Four of the patients were female and one male. All were suffering with intense agonizing pain in the epigastric region, sitting up in or on the edge of the bed. Examinations showed marked tympany over the stomach, varying degrees of shock, fixed attitude and tremendous anxiety. The abdominal wall in each case was tense and the character of pain might easily have been confused with that caused by ruptured peptic ulcer, because of its severity, sudden onset and location. Tenseness of the abdomen differed from that of ruptured peptic ulcer, because of the fact that it was slightly more yielding to palpation and not of such rigid board-like nature as that caused by the latter condition. Five minims of 1-1000 solution of adrenalin were given by hypodermic in every case and this produced marked relief of the spasm in the females within five minutes. In the male three doses were required at five-minute intervals before the spasm began to subside. Only one case out of the five has had a recurring attack and this was in a young woman, aged thirty-four, first seen on September 24, 1937, who was completely relieved at the time but had a recurrence seventeen days later, on October 11, 1937, while waiting for me in my office, which attack required three injections. Under medical care she has gained nine pounds in weight and has correspondingly improved in general health.

Acute pain from peptic ulcer due to local reflex spasm in the muscle fibers underlying the ulcer might be relieved and perforation prevented by the hypodermic use of adrenalin.

It will be readily seen that, had any of these cases been perforations, adrenalin would have been of no avail.

The use of adrenalin was prompted by my appreciation of the similarity of the neurophysiology involved in spasm occurring in the digestive tract and of that in bronchospasm as referred to above, and its use involved no risk and caused no delay, had these cases been ruptured peptic ulcer.

In discussing Dr. Adams'[7] paper, entitled "Factors in the Etiology of Peptic Ulcer", at the Tri-State meeting held at Norfolk last February, and published in *Southern Medicine and Surgery* of May, 1937, I emphasized the trophic effect upon the stomach wall produced by vagal stimulation predisposing etiologically to the formation of ulcer. Irritation resulting from prolonged spastic conditions might cause inflammation and contribute to formation of ulcer which, in turn, might further stimulate and irritate the vagus, thus maintaining spasm to continue a vicious cycle in the ulcer.

For intelligent treatment after the pylorospasm

has subsided, study must be made to determine factors which are disturbing the autonomic nervous system.

SUMMARY

1. Acute pylorospasm, because of similarity of acute symptoms to those of ruptured peptic ulcer, might mislead us into having our patients operated upon unnecessarily;

2. Acute pylorospasm can be relieved by one or more hypodermic injections of adrenalin 1-1000 at 5 minute intervals, 5 minims in each dose;

3. Loss of time is negligible and should ruptured ulcer exist no harm is done;

4. The physician who thus prevents unnecessary operation gains the gratitude of his patient;

5. This treatment may prove of value in relieving gallstone colic.

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INSULIN SHOCK TREATMENT OF DEMENTIA PRAECOX.*

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Until quite recently dementia praecox, the dreaded adolescent disease, was considered a hopeless condition. Very little thought has been given to treatment but much time has been consumed diagnosing and classifying, and when this is finished, medical science discontinues its endeavors and the unfortunate victim becomes one of the 150,000 dementia praecox patients who are receiving custodial care in our state hospitals. At the present time many of our hospitals are enthusiastically treating dementia praecox by one of the several newly designed methods, namely, insulin shock treatment, camphor[15] and metrazol.[13] Insulin shock therapy, originated by Dr. Sakel of Vienna in 1933, is most widely used and has been used in this country for about eighteen months. Results have been encouraging—with remissions three or four times greater than can be expected from spontaneous remissions. No one can foretell the ultimate outcome of this therapeutic enthusiasm but certainly this general change in attitude toward dementia praecox should be a progressive step in the solution of this mystery of medicine.

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The purpose of this paper is to better acquaint the physicians of the state with the insulin shock or hypoglycemic shock treatment of dementia praecox. Our information has, been derived from a review of the literature, a short period of observation at Bellevue Hospital, and the treatment of six cases. A description of the treatment, theories of the treatment and general results will be briefly considered, followed by our case reports.

The treatment consists of a progressive insulinization of the patient by the intramuscular administration of insulin in gradual increasing doses until the shock stage is reached. The patient is then allowed to have daily shocks with one day of rest weekly until satisfactory improvement occurs or the treatment is given up. In describing the *treatment,* Sakel divides the procedure into *four phases.*

Phase 1.—The *Introductory Phase* is started by giving the fasting patient 5-10 units of insulin between 7:00 and 8:00 A.M. The patient is kept in bed and four to five hours later is given sweetened orange juice. The mid-day meal follows and then the patient is allowed to carry on the usual hospital routine. The insulin is increased 5-10 units daily

until the shock dose is reached. During this period hypoglycemic manifestations occur with increasing severity. They consist of hunger, profuse perspiration, drowsiness, somnolence, increased flow of saliva, tremors and muscular twitchings.

In debilitated cases we feel it is best to start Phase 1 by giving small doses of insulin about two hours before each meal. The doses can be gradually increased and by so doing the patient's tolerance to insulin is discovered. Immediately the patient begins to gain weight, is less restless and shows general physical improvement. This method may be prolonged for two weeks if necessary before starting the early morning dose followed by the four to five hour fasting period.

Phase 2.—The *Shock Phase* is the most important stage in the treatment. This stage is assumed to have been reached when the dosage of insulin produces a degree of coma which no longer enables the patient to drink a sugar solution and tube feedings have to be resorted to. The shock dose of insulin varies; a few cases have had severe shocks on 20-30 units, while other cases require over 300 units for a severe shock, the average shock dose being 60 to 100 units. The coma, or true shock, comes on two to four hours after the injection. A patient is considered comatose when the corneal reflexes have gone and in very deep coma when the pupils fail to react to light. During the shock phase the normal reflexes disappear, the Babinski sign and ankle clonus become positive. Occasionally after a severe shock a hemiplegia or aphasia of a few hours' duration is observed. Some patients are very restless, throw themselves around in bed and require much care to prevent injuries. Many patients have muscular twitchings and myotonic attacks. Grand-mal epileptic convulsions are not uncommon and the treatment is terminated immediately after such an attack. Some feel that convulsions are a bad prognostic sign and others feel they are beneficial. Convulsions are more common in patients who do not perspire freely. The pulse is usually slow during shock and the temperature often drops several degrees due to the profuse perspiration. Blood sugar readings usually range between 40 to 0 during this stage but have no particular significance.

The second phase is terminated after a period of coma of one to two and one-half hours. The best method is the giving of 150 to 200 gms. of dextrose in fruit juice or water by means of a nasal tube. We have found Karo syrup to be the most satisfactory form of sugar. One must be very careful that the tube goes to the stomach. If any fluid can be withdrawn, it should be tested with litmus and air should be blown into the stomach which can be heard entering the stomach with a stethoscope on the abdomen. Patients usually come out of shock ten to twenty minutes after the tube feeding. Some patients are very noisy and struggle considerably while coming out. Special indications for termination of shock are: Pulse above 120 or below 40, convulsions, impairment of respiration, laryngeal spasm and unusual pallor or cyanosis. If an emergency situation arises 1 cc. of adrenalin should be given hypodermically followed by 50 per cent glucose intravenously, and after this procedure the patient should drink a sugar solution or have it introduced with a tube.

Phase 3.—The *Rest Phase* is nothing more than the routine one day of rest each week on Sunday and any other additional days of rest that are deemed wise for the patient's welfare.

Phase 4.—Stabilization Phase is carried out by giving a smaller dose of insulin and interrupting the hypoglycemic period while the patient is able to drink the sugar solution. It is supposed to consolidate and stabilize the gains acquired during Phase 2. This phase is not considered important and very little time is used for it.

Practically all cases treated receive some beneficial results: Physical improvement and sedative effect, if nothing more. Many cases who do not have remissions become less disturbed, are easier to handle and are more sociable in the hospital. If the patient shows the most desirable response his progress will be as follows: At first he becomes lucid and exhibits normal behavior traits during hypoglycemia especially during the period immediately preceding coma. Later on the most marked improvement is noticed just after the termination of shock, and he may be symptom-free for a short period. The symptom-free period increases in length until the patient is symptom-free all day but may show some reactivation of old symptoms during hypoglycemia. This phenomenon is called "the reversal of reaction". As the patient improves more he shows fewer psychotic symptoms even during hypoglycemia. When this stage is reached the treatment is concluded after a few days of Phase 4 treatment. Some cases have been carried on for six months but the most suc-

cessful cases have been those who did not require more than four to six weeks of treatment for a complete remission.

The paranoid, catatonic and stuporous cases should be handled differently for best results. The paranoid case has the best prognosis and when improvement occurs it is progressive and orderly. Deep coma is best for these cases and the hypoglycemia should not be interrupted during the stage of activated psychosis. In catatonic cases it is wise to interrupt the hypoglycemia before actual coma is reached. In stuporous cases interruption during an activated state is desirable.

Any therapeutic procedure as severe as insulin shock treatment must be considered dangerous. However, the mortality has not been high; of the first 300 cases reported in Europe there were four deaths. Seventy cases have been treated in Bellevue Hospital with no mortalities. The patient runs a risk as in a surgical operation, and as in a surgical operation the possible gains over-shadow the dangers. The patient must have careful nursing care during the treatment. After-shock may occur as late as twelve hours after treatment and if it occurs the nurse should be able to recognize it and treat it by giving the patient a sweetened drink. The heart must be kept in mind, but so far no serious heart complications have been reported. Aspiration of saliva resulting in aspiration pneumonia is one of the most dangerous complications. The patient should be kept on his side with head placed so that the saliva will run out of the mouth. Atropine is indicated in some cases. Extreme care should be exercised during the tube feedings.

The universal question on hearing of insulin shock therapy for the first time is, what is the rationale of the treatment; why and how does it work? No one can answer this question, but several of the theories will be mentioned: (1) Sakel favors the following theory, briefly stated: Insulin inhibits that which is functioning, meaning the psychosis, and activates that which is latent, meaning normality. (2) The shock theory: Sakel says, "Perhaps through the elementary convulsion of the cell, almost amounting to its destruction, every conduction-path not yet firmly fixed may be destroyed and on recovery the original normal conduction-paths predominate and can easily be polarized in the normal direction through further treatment." Professor Berze[16] says, "Probably nothing more is concerned than psychic trauma or emotional shock bound up with insulin treatment." This shock theory can not be applied to all cases because some cases show definite improvement before a stage of shock is reached. (3) Metabolic theories: Sakel speaks of a detoxicating effect on the body as a whole by influencing the basal metabolism. Several workers have suggested that insulin therapy improves the nutrition of the brain because of its stimulating effect on carbohydrate utilization and since it is known that brain tissue is directly and exclusively dependent on carbohydrates for nutrition.

TABLE NO. 1

Clinical Results of Insulin Therapy in Dementia Praecox.

Source	No. Cases	Full Remission	Improved
Vienna (Sakel) _____	104	45%	21%
Switzerland (Muller)	70	50%	27%
New York State Hospitals _____	89	32%	41%
Bellevue Hospital____	30	37%	33%

As a small percentage (5-25 per cent) of cases of dementia praecox are known to undergo spontaneous recovery, it is somewhat difficult to evaluate the results of treatment. However, the results achieved by insulin shock far exceed any that might be expected to recover spontaneously.

Table No. 1 is a summary of reported results in 293 finished cases. "Full remission" means no symptoms, full insight and able to work. Under the "improved" heading is included patients who are at home and working, but not entirely well; and cases who are improved but still require hospitalization.

The results as tabulated in the above table include old and new cases. Of Sakel's new cases, i.e., those who have been sick less than six months, he reports 70 per cent in full remission and 16 per cent improved. His old cases, or those sick for a period of six months or more, he reports 20 per cent in full remission and 45 per cent improved. Muller's results are approximately the same as Sakel's when sub-divided into new and old cases. The New York State Hospitals report includes only old cases who offered a poor prognosis with little hope of spontaneous recovery. A number of the Bellevue patients were new cases.

From all reports, we can safely conclude that from 60-70 per cent of the cases of dementia praecox under six months' duration have responded to insulin therapy with full remissions. Very few full

XUM

remissions have been obtained in patients who have been sick over one and one-half years.

The remissions have been fairly stable. At the end of Sakel's first two years of work, he had fifteen remissions lasting over one year, and thirty-four over six months. There were eleven relapses, five of which were again treated and with good results in three cases. Some of the Bellevue cases have been at home for six months with no relapses. Sufficient time has not elapsed to make a statement as to the permanence of the results.

TABLE No. 2

Clinical Resumé of Insulin Shock Cases.

Case	Type	Duration	Shocks	Dose	Results	Remarks
1.	Deteriorated	6 yrs.	16	40 U	Unimproved	In State hospital.
2.	Paranoid	4 mo.	12	50 U	Well	At home 3½ mo. at work.
3.	Hebephrenic	1 yr.	20	55 U	Unimproved	Marked physical gain.
4.	Paranoid	2 yrs.	12	70 U	Improved	At home 2½ mo. at work.
5.	Hebephrenic	2 yrs.	16	40 U	Improved	Still under treatment.
6.	Hebephrenic	4 mo.	20	100 U	Improved	Still under treatment.

CASE HISTORIES

Case 1.—This patient, a woman, aged thirty-one, began showing symptoms of dementia praecox six years ago and has had institutional care for four years. She has shown progressive mental deterioration. The insulin shock therapy was started April 6, 1937, at the request of the family. She had sixteen moderately severe shocks with the injection of forty units of insulin. We did not notice any beneficial effects, either physical or mental. She is the only case in our series that has not shown some response to insulin therapy.

Case 2.—Patient, man, aged twenty-nine became restless and was unable to concentrate on his work in February, 1937. The nervous state gradually progressed and on March 30 he had to be transferred to a state hospital. He felt that he should save the world and that he was being persecuted. He endeavored to carry out the commands of imaginary voices and in doing so was frequently extremely violent. A diagnosis of dementia praecox, paranoid type, was made at that time, which was later confirmed by an independent examiner, prior to his admission to the hospital May 23. At this time he was a very dangerous patient, both to himself and to others. Insulin shock was started May 28 and completed June 28. After six days of treatment, before the shocks developed, he began to improve and to gain some insight into his condition. He received twelve wet shocks on fifty units of insulin. The improvement was continuous and progressive. Two weeks after beginning the treatment, the family considered him normal from the mental standpoint. He gained complete insight into his condition and gave a very remarkable account of his illness with full explanation of his psychotic actions. During the treatment he gained thirty pounds in weight. He was discharged from the hospital July 1 and resumed his duties as foreman in a textile plant, which position he has held for the past three months. He is considered perfectly well.

Case 3.—Patient, girl, age eighteen, had suffered from a mental illness for one year prior to her admission May, 1937. During this time she had been seclusive and refused all foods except fluids. She was brought to the hospital by ambulance because of irrational behavior, silly delusions and self-imposed starvation of three weeks' duration. At that time she was in a stuporous state, arousing at intervals to talk of her delusions in an excited manner. She showed marked mental regression, was untidy and had to be tube fed. She had lost forty-five pounds during the last year, weighing seventy-one pounds on admission. Insulin shock therapy was begun June 9 and completed July 23. During this time she had twenty shocks with fifty-five units of insulin. The patient had several grand-mal convulsions during the shocks. After a few days of treatment she began to eat and gained rapidly in weight. Marked mental improvement was noted after six or seven shocks. The improvement was not continuous or progressive but at one time she was well enough to act as hostess at a hospital tea. For a week prior to discharge, she was communicative, interested in her appearance and enjoyed walking on the grounds with a nurse. She never realized her true condition. She gained sixteen pounds during the treatment. After a few days at home, she became worse and was taken to a state hospital. It is probable that her recovery might have been permanent had she received adequate treatment. The

response of this patient to insulin shock was definite physical improvement with frequent periods of approaching-normal mental functioning.

Case 4.—This patient, a woman, aged forty-five, had been suspicious of her husband and neighbors for several years. For about one year before admission she had made life almost unbearable for her husband, accusing him of unfaithfulness, believing her minister and other good friends were making remarks detrimental to her character. She felt that others were in sympathy with her husband and against her. Her husband brought her to the hospital because her increasing delusional state made it impossible to keep her in the home longer. She was in the hospital six weeks before insulin treatment was begun. During this time she became gradually worse, involving the hospital personnel in her delusional pattern. Before the shock stage was reached, a general sedative effect was noticed. The shock dose was seventy units and she received twelve wet shocks without reaching the stage of true coma. It was noted that in the afternoons following the shocks, she was quite sociable and open in her conversation and did not discuss her various delusions. She was happy, slept well and had a normal interest in life. After reduction of insulin, in the fourth stage, her old symptoms reappeared in a mild form. She has been at home for the past two months and has been able to care for her home. Although she cannot be considered well, she is much better than before receiving the treatment. We feel that a longer course of treatment and deeper shocks would be beneficial in her case and likely result in a complete recovery.

Case 5.—Patient, man, aged twenty-one, has not been well for two years. His first symptoms were associated with his stomach and later a feeling of depression, indifference and restlessness. In June, 1937, he became so restless and so peculiar in his behavior that he was sent to a state hospital. He was admitted July 10 and at that time was quite restless, perplexed, preoccupied, apathetic, grimaced and had many mannerisms. He was too preoccupied to converse. He had lost fifteen pounds in weight. During the first two weeks in the hospital he showed no improvement. Insulin therapy was started and he began to improve immediately. He developed a hearty appetite, was less restless and began sleeping without sedatives. We gave him sixteen moderately severe shocks which required forty-five units of insulin. After the sixth shock and until his discharge two weeks later, he showed no abnormal behavior and was fairly sociable. He had normal interests and conversed intelligently and showed very little preoccupation. There was a gain of fifteen pounds in weight. He did not, however, develop insight into his condition. After the sixteenth shock he was sent home for a trial and remained in his improved state for three or four days. He did not maintain his improvement and has returned for additional insulin shock therapy.

Case 6.—This patient, a boy, aged sixteen years, has been physically weak and a problem child all his life. An examination by Dr. Howard R. Masters, three years ago, showed "a schizophrenic personality". Definite psychotic symptoms were first noted in May, 1937, at which time he injected household ammonia under the skin of his left arm with suicidal intent. In a few weeks he refused to talk or eat and was sent to a state hospital. He was in the state hospital for three months during which time he became untidy, was mute, and had to be forced fed. These symptoms were verified by our study of his case for one week before starting insulin therapy. Four days after beginning insulin, he began feeding himself and in a few days he talked, associated with other patients and became tidy. All these changes occurred before a shock dose was reached. He has received twenty shocks thus far on 100 units of insulin each morning. He is showing evidence of a gradual integration of personality. He has gained ten pounds in weight. We do not anticipate a full recovery in this case as the patient is a defective boy mentally and physically. It is likely that the improvement will be sufficient for him to be cared for at home. He is still under treatment.

CONCLUSIONS

We are convinced that there is a definite relationship between improvement of dementia praecox patients and the insulin shock treatment. One must witness the marked change these patients undergo before fully appreciating the value of this treatment. It is without question the best available therapy at this time.

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DISCUSSION

DR. D. C. WILSON, Charlottesville: Dr. Strickler presented a splendid paper and his discussion of his cases is very clear and well done. Insulin treatment of dementia praecox is still in the beginning stage. At the present time the New York State Mental Hospitals have taken over this therapy and are going to use it on a great many patients in their efforts to try out its value. Certainly if these people have remissions as long as six months to a year, the treatment will be worth doing. At the present time at the University Hospital we have several cases under insulin shock therapy and they have improved. We also now are using metrazol to produce convulsions and with these we have had more success than with the insulin. I was struck by the fact that Dr. Strickler claimed shock reactions with relatively small doses. Usually we have had to give as high as 100 to 125 units to obtain shock. I have noticed in some of the cases from up north that they have used as much as 450 units of insulin in one day. We still do not know which cases should be treated and whether a patient with a poor pre-psychotic history should be treated with insulin shock or not. It has been claimed by some that some individuals treated are better than they ever were. There is some possibility of improving people who have been mentally sick for a long time without its being recognized. We have yet a great deal to learn but undoubtedly Dr. Strickler has gone ahead with his work in a well-controlled style. Certainly we must be careful with our conclusions and very careful in our treatment of these people so that we do not do more harm than we do good. Certainly it is a form of therapy to be used in institutions and to be carried out by people who are trained and careful in their work.

DR. ASA SHIELD, Richmond: Insulin treatment has been used long enough to say that this contribution of Dr. Sakel's in 1933 is now generally recognized as the most effective therapy in the treatment of dementia praecox. As our State society is essentially one of practicing clinicians, it is interesting that this very important therapy is the contribution of a clinician who observed that disturbed personalities in morphine withdrawal and certain psychotic manifestations were quieted following insulin therapy and that some of the psychotics seemed to go more quickly into remissions following this therapy.

Dr. Sakel's original cases were very carefully checked and the diagnosis was confirmed by his chief, Dr. Potzol. In Vienna the criterion for making the diagnosis of dementia praecox was more in line with the Kraeplin school, so I think it is fair to assume that the more malignant dementia praecox cases were treated there than in some places where the diagnosis of dementia praecox is a label used for a broader group of personality disturbances.

It is of interest that when this work was first being done and reported, Dr. Joseph Wortis, of New York, was in Vienna, at which time he translated one of Sakel's reports and sent it to various medical journals in America; it was a long time before this report was published.

We may ask, what can we expect from insulin therapy? It is generally accepted that about 30 per cent of the first admissions of dementia praecox return home. With insulin

therapy various conservative clinics have reported that 70 to 79 per cent of the cases who had been ill for only a few months recovered sufficiently to return home. This is very much in line with Dr. Strickler's observation that a patient who had been ill for four months got the best results and this is confirmatory of our general feeling that it is advantageous to get these patients early, as the outlook for remissions (that is all we can say at this time) is better in these cases.

The convulsive therapy as first reported in 1934 by Meduna, of Budapest, has recently been reported in a monograph, with results as favorable in cases of dementia praecox of only four or five years' duration. Insulin therapy has given a few favorable reports in cases with longer duration, which has surprised Dr. Sakel and others.

Dr. Wilson stated in his discussion that he preferred to have his patients in shock about four hours. In the observation of my own cases and those I saw in other clinics two hours was considered the maximum shock period. In all of my cases except one that went into an irreversible coma the termination of the insulin stupor has depended on, first, the clinical manifestation of the illness in association with the manifestation and the stupor when there were no untoward signs developing such as collapse, low temperatures, and profound muscular rigidity with convulsions.

We have been impressed with the fact that our patients who have convulsions seem to respond to therapy better, and, contrary to what some of the discussions seem to indicate, we rather like to see them as long as the convulsions are not associated with any complications.

It is of further interest to note in reports of institutions where a large number of cases have been treated that the results seem to be consistently more effective; thus, I think this is a therapy that in proper hands offers something of real value, whether temporary or not, to both the patient and the family of those who have dementia praecox.

DR. REX BLANKINSHIP, Richmond: My experience with insulin therapy has been rather limited but to me it has been just about as shocking as it has been to the patients. I have had some dramatic reactions and have felt very discouraged by the results I have had in my two cases. Of course, that is a very small number of cases but there were not any encouraging features at all.

Last year at the American Psychiatric meeting in Pittsburgh, I was very much amazed to see the enthusiasm that all the older men had for this treatment. I felt much, as Dr. Shield did, that perhaps it was a reaction to the psychoanalytical advocates.

Last fall, while visiting in New York at the Hudson River State Hospital, where they were at that time running a series of insulin shock cases, they also reported some controls. These controls were given practically the same treatment, care and attention, but no insulin was given. It seems quite definite that there must be some psychological reaction in these patients due to the increased attention received during treatment.

I enjoyed Dr. Strickler's and Dr. King's paper very much.

DR. STRICKLER, closing the discussion: I enjoyed the discussion very much and wish to thank the gentlemen for discussing the paper.

Dr. Blankinship remarked about the controls. I have not read the article he referred to, but I have read some reports along that line. I know it has been said that increased attention might be the explanation. One writer, while doing experimental work with a group of dementia praecox patients, did not feel that the extra attention given them resulted in any definite improvement.

ACUTE HEAD INJURIES—WITH PARTICULAR REFERENCE TO TEMPERATURE, PULSE AND RESPIRATION CURVES.*

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The diagnosis and treatment of acute head injuries has become an increasingly important problem in modern surgical practice. These injuries may be classed as surgical emergencies and they are of interest not only to the specialist in neurologic surgery but to every practitioner of medicine and surgery. Their number is increasing yearly, due to the steady advance in the number of vehicular accidents. Finally, they command our attention because there still exists considerable difference of surgical opinion concerning the proper procedures to follow in the diagnosis and treatment of such injuries.

The advent of modern collision forces has produced more diffuse and more formidable injuries to cerebral tissue than we have routinely experienced heretofore. Such injuries have rightly emphasized the intracranial disturbance and such a phrase as "fracture of the skull" should be discarded from our surgical thinking. Acute head injuries threaten the preservation of life and only in rare instances does an appreciation of the integrity of the skull influence our treatment. The presence of foreign bodies or the clinical impression of a depressed fracture ob-

*Read before the Lynchburg Academy of Medicine, December 6, 1937.

From the Division of Neurosurgery, Duke Hospital, Durham, N. C.

XUM

viously demand roentgenographic study. We are interested in the question of fracture lines crossing the paranasal sinuses, the mastoid cells, or the region of the middle meningeal artery. It must also be remembered that the presentation of a skull roentgenogram in a court of law sometimes carries much more weight than any assertion by a surgeon concerning the degree of intracranial injury. Except in exceptional circumstances, however, immediate roentgenographic study of the skull is not indicated in acute head injuries where every unnecessary manipulation is contraindicated.

Throughout the history of surgery, we can find enthusiastic individuals advocating some type of specific routine in the diagnosis and treatment of acute head injuries. Early in the nineteenth century, for instance, Percival Pott trephined all head injuries and he would frequently follow a linear fracture line across the skull with as many as twenty trephine openings. Indeed, a member of the royal family gave him a testimonial as a tribute to his dexterity. And at the same time, Charles Bell, of Bell's palsy fame, advised non-intervention, nursing care and like conservative measures. Similar differences of opinion are present among members of our modern surgical family. Early in the present century, subtemporal decompression was introduced in the therapy of head injuries[1] and the resultant mortality of ill-chosen operations was appalling. Following the work of Weed and McKibben[2], who showed that intracranial pressure could be reduced with the introduction of hypertonic glucose solution, it was not long before we found such therapy well established in the clinic[3]. This was soon followed by the dehydration or restriction of fluid type of therapy[4]. The so-called medical treatment of head injuries was rounded out by the application of lumbar drainage through repeated lumbar puncture[5]. Study of hypertonic glucose therapy disclosed the fact that following the initial fall in pressure[6], there was often a delayed rise in pressure greater than that existing previously, probably due to the irritative effect of the solution upon cerebral tissue. Sucrose in the same percentage solution has been found to be a more satisfactory substance, lacking any irritative effect[7].

I have emphasied these therapeutic measures that exist today in order to demonstrate the belief that the therapy of head injuries has become more active and that this activity has shown a tendency to fall into a definite routine. Head injuries are being treated as a group rather than as individual problems which they actually are.

To retrace our steps a bit, how are the more diffuse head injuries diagnosed? It is obvious that individuals with local destruction of skull and cerebral tissue, and those with focal neurologic deficit offer little difficulty. But in this group, and in those individuals with more diffuse injuries, we wish to know how much intracranial pressure exists and we wish to follow the fluctuation of that pressure. There has been and is a growing tendency to use spinal puncture with study of the spinal fluid and direct measurement of the subarachnoid pressure as agents for the estimation of the extent of injury. There are three obvious disadvantages to this procedure. Quantitative study of the red blood cell content of the spinal fluid bears no clear-cut relationship to the severity of the cerebral injury. Accurate spinal fluid dynamic studies are extraordinarily difficult to obtain upon many patients, especially those who are restless or disoriented. In the third place, spinal puncture, even if carefully done, may give further room for hemorrhage if bleeding from an extradural, subdural or basilar source be present. In patients with acute subdural hygroma, no information of value may be obtained from lumbar puncture.

In our differential diagnosis we must place, on one side, cerebral edema of varied degree, and, on the other side, space consuming lesions such as extradural hematoma, acute subdural hematoma and acute subdural hygroma. All have in common the factor of increased intracranial pressure. Our problem, reduced to the simplest formula, is to recognize and evaluate the amount of increased intracranial pressure and then to rule out the above individual syndromes that demand so-called radical or operative intervention to preserve life. The remainder may then be treated with the so-called conservative methods, i. e., intravenous sucrose, dehydration, and spinal drainage, followed by subtemporal decompression if necessary. Many of these will need no treatment although dehydrating methods may reduce subjective discomfort.

How may we answer the first part of the problem outlined above? Of paramount importance is the state of consciousness of the individual patient. A definite march of the state of consciousness may be observed in individuals with increasing intracranial pressure. One observes first drowsiness from which

the patient may be easily aroused. This early dulling of the sensorium may be marked by the onset of involuntary micturition or defecation. As pressure increases restlessness may appear with periods of alternating hyperactivity and semicoma. This phenomenon is of serious portent. The periods of semicoma may rapidly increase in length until full coma is established. Very often this march in the state of consciousness is reversed as patients recover following relief of intracranial pressure. Individuals may at once enter any of the above states of consciousness following head trauma and our observations must be directed toward the recognition of what might be termed adjacent states of consciousness. Unless we know that intracranial pressure has been relieved or is receding, all medication is contraindicated for fear of concealing these important diagnostic signposts.

There are three important clinical observations, namely, those relating to the temperature, pulse and respiration curves, that, with the state of consciousness, will answer the first part of our problem in every case. It has been taught for many years that a bradycardia represents a state of compensated intracranial pressure, that a break in this bradycardia, an irregular pulse, a rise in temperature and a high or falling blood pressure represents an impending collapse of an individual's resistance to intracranial pressure. It is not quite as simple as this but published studies of 300 cases[8] and unpublished studies of a similar number of more recent cases have suggested that these clinical observations may form the basis for a valid estimation of the degree of intracranial pressure.

The study of such clinical charts is based on the fact that trauma to the brain results in the same sequelae as elsewhere, namely, edema and hemorrhage, with or without local destruction of tissue. When the brain, in its enclosed, rigid skull, can no longer compensate for an increase in volume, by decrease in the vascular bed and the ventricular space, the medullary centers are affected. Such effects are reflected in temperature, pulse and respiration charts.

It has been stated as a general principle, and correctly so, that 70-75 per cent of acute head injuries will survive, with or without treatment; that 15-20 per cent will need some type of intervention to preserve life, and that 10-15 per cent will succumb to the injury no matter what treatment is instituted.

Individuals in this first, and numerically the largest group, show a clinical chart that has been previously described as Type I.[8] It is characterized by an initial loss of consciousness, followed by a return to complete consciousness or a varying period of drowsiness. The temperature curve, after an early drop or after starting from a normal level, shows a mild hyperpyrexia to 101 degrees rectally and a slow, regular decline to normal within from twenty-four to sixty hours. The curve for the pulse rate closely follows the temperature curve, rising steadily with it after an initial irregularity and falling to normal in the same length of time. The respirations are normal, or during the period in which the temperature rises, they may show a slight but regular retardation. The patients in this group fall into that class that has been termed "cerebral concussion". It is obvious that the surgeon has no active part to play in these injuries except that of closely observing the clinical course and possibly treating local scalp lacerations or reconstructing a depressed skull. The compensatory mechanism of the brain is able to control the mild degree of intracranial pressure. Late in the course of the injury, subjective symptoms may safely be relieved by dehydration methods.

Acute head injuries that fall into the next two groups by virtue of a state of consciousness and representative charts for the temperature, pulse and respiration curves are the most interesting and the most amenable to surgical intervention when the natural defenses of the body collapse. The initial loss of consciousness is more profound and more prolonged. Throughout the course of the injury, there is a more extensive loss of consciousness, varying from drowsiness to restlessness and periods of hyperactivity contrasting with semicoma. Most characteristic of this type is an initial bradycardia, with a persistent, moderate rise in temperature. The slow, heavy, pounding pulse may develop late, from an extradural hematoma for instance. Such a bradycardia is a potentially dangerous manifestation and is not necessarily evidence of compensation. The respiratory rate tends to become more irregular. The third group, or type, presents the same picture in a more aggravated form. The temperature is high and unremitting, there is a relative bradycardia and the respirations show a tendency to become Cheyne-Stokes in character.

The fourth type includes those acute head injuries of such a degree that no procedure is successful in

XUM

nullifying the cerebral damage. It is clearly signalized by immediate and lasting coma, a rapidly rising pulse rate and temperature and a Cheyne-Stokes type of respiration. Representative charts are included to illustrate these four types of injury. It might be said in passing that similar charts are used in my clinic for the pre- and post-operative observation of cerebral tumor patients and have been found of similar value in the estimation of intracranial pressure. We have felt that blood pressure readings lag behind these other clinical manifestations of pressure and we use the estimation of blood pressure solely to determine the existence of surgical shock.

Having defined the clinical charts of Type II and III acute head injuries as those representing degrees of increased intracranial pressure that are potentially dangerous to the preservation of life, how may we determine the optimal period for intervention? A low percentage of individuals with absolute bradycardia will survive without intervention. Unhappy clinical experience has demonstrated the fact, however, that such individuals may rapidly "de-

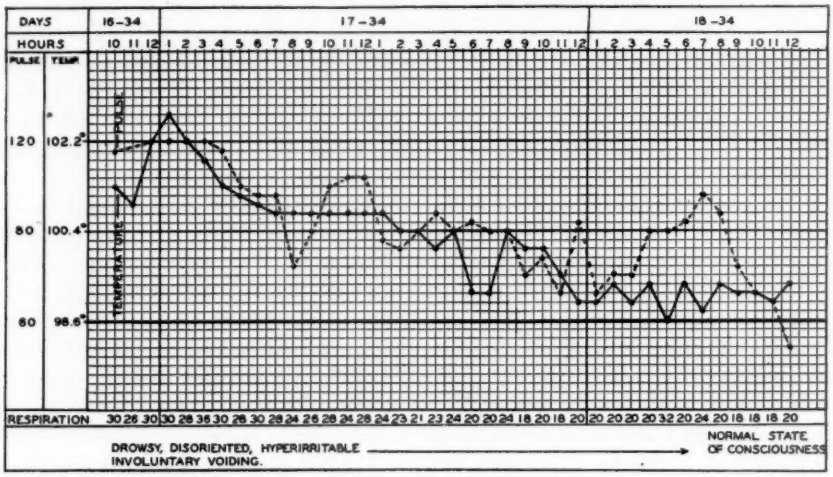


FIG. 1. CLINICAL CHART OF TYPE I INJURY.

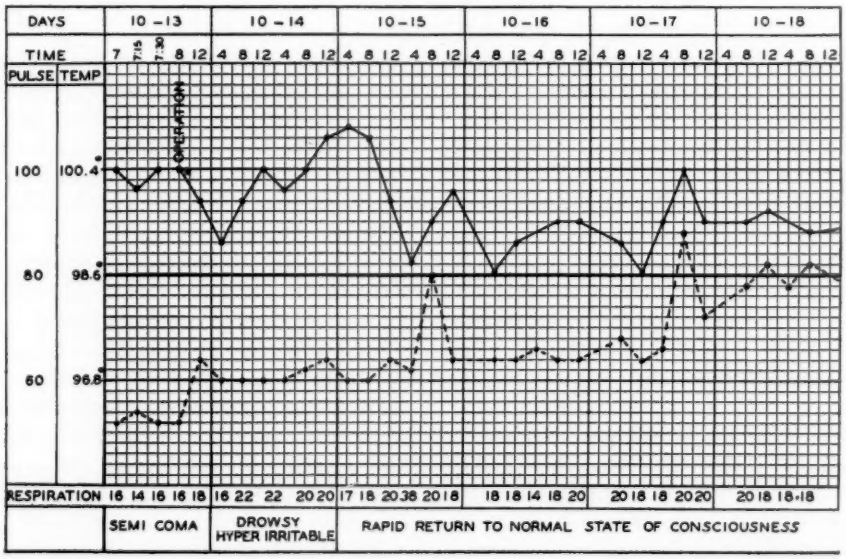


FIG. 2. CLINICAL CHART OF TYPE II INJURY. THIS PARTICULAR CASE IS AN EXTRADURAL HEMATOMA.

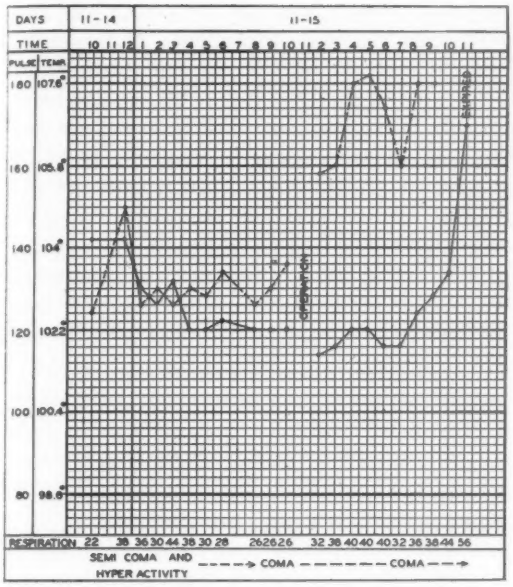


FIG. 3. CLINICAL CHART OF TYPE III INJURY, UNRELIEVED BY SUBTEMPORAL DECOMPRESSION AND DEHYDRATION

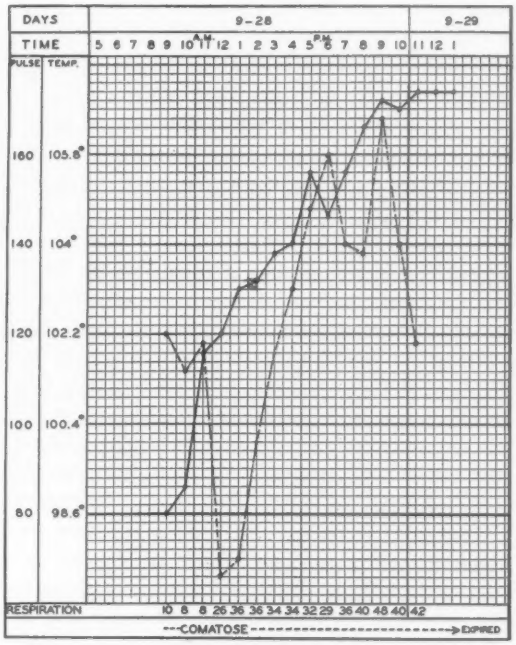
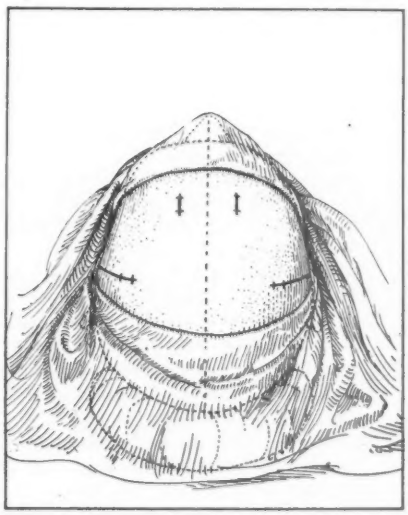
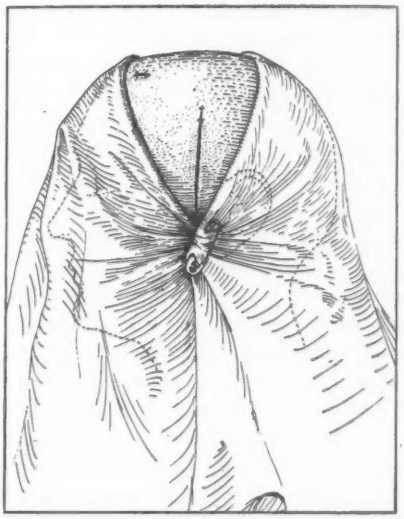


FIG. 4. CLINICAL CHART OF TYPE IV INJURY.

compensate" and die without any further change in their clinical charts. As signs of decompensation, we would watch for—1, deepening of the state of consciousness; 2, deepening or the slightest irregularity in the absolute or relative bradycardia; 3, the slightest rise in temperature, and, 4, irregularity in the



PATIENT LYING ON BACK. HEAD DRAPED FOR BILATERAL FRONTAL AND TEMPORO-PARIETAL PERFORATER OPENINGS AND FOR BILATERAL SUBTEMPORAL DECOMPRESSION.



HEAD MAY BE ROTATED TO EITHER DIRECTION DEPENDING UPON THE SIDE CHOSEN FOR DECOMPRESSION

FIG. 5. METHOD OF DRAPING PATIENTS REQUIRING OPERATION FOR ACUTE HEAD INJURY. FRONTAL TREPHINE OPENINGS MAY BE USED FOR LAVAGE OF BOTH ACUTE AND CHRONIC SUBDURAL HEMATOMAS AS DESCRIBED IN THE TEXT.

XUM

respiration rate. Ideally, treatment in these types of acute head injuries should be anticipatory, namely, before signs of decompensation are manifest.

Three sequelae of acute head injuries must be ruled out immediately in individuals demonstrating clinical evidence of increasing intracranial pressure. All three threaten the preservation of life and their non-recognition is a tragic circumstance.

Extradural hematoma may be seen with the usual clinical history and picture of trauma to the head, with initial loss of consciousness, a free interval followed by progressive coma, convulsions in the contralateral face and arm, and, finally, paralysis developing in the same sequence. Unfortunately, the clinical history may not be obtainable and patients may show only the evidence of increasing pressure[9]. Acute subdural hematoma characteristically shows no well-defined syndrome except that of pressure[10]. The same may be said of acute subdural hygroma[11], a collection of fluid entering the latent subdural space through arachnoid perforations. The operative diagnostic and therapeutic attack portrayed here is simple in execution and may be done under local anesthesia, with avertin supplement if necessary. The accompanying illustration shows the patient draped so that a diagnostic perforator opening may be made in either temporo-parietal region, according to possible localizing signs or on both sides in individuals presenting only evidence of increased intracranial pressure. Extradural blood clot is at once apparent and a subtemporal decompression on the indicated side with ligation of the middle meningeal artery may be carried out. Simple puncture of the dura may evacuate a collection of subdural fluid or blood. If actual blood clot is present subdurally, a small decompression opening may be made for removal of the lesion. Bilateral anterior perforator incisions are routinely marked out so that a second perforator opening may be made to facilitate lavage of a hematoma. Such perforator openings are routinely used in the treatment of chronic subdural hematoma. If nothing is found, we know that we are faced with the problem of combating uncomplicated cerebral edema. Whether to proceed with subtemporal decompression at this point is a matter of individual surgical experience and preference plus the exigencies of the individual case. The operative intervention described above adds but little to an already over burdened patient compared to the relief that may be obtained. If no specific lesion is disclosed by this procedure, dehydration therapy is essayed, using sucrose 50 per cent intravenously in amounts depending upon the age and therapeutic response obtained. The initial treatment is 200 cc. of the solution, given over a period of thirty minutes, repeated every four to six hours, together with restriction of fluid intake to 1200 cc. a day for an adult individual. Early failure of response to increasing amounts of such hypertonic solution is an indication for subtemporal decompression in the classical method.

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REMARKS ON EVIPAL ANESTHESIA.*

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Evipal soluble, a general anesthetic, is a chemical of the barbituric group. Its name is long; we cannot spell or pronounce it. More than one hundred papers and monographs during recent years have been published reporting upwards of thirty thousand cases in which evipal has been used. In only nine instances was this an unsatisfactory anesthesia.

We are sure that many of you present have read these reports; therefore we will refrain from quoting, but will speak as if a résumé of the same.

Complete anesthesia is had in about forty seconds. The blood pressure usually falls about 15 mm. Hg., returning to its former level on awaking. The pulse rate and character usually remain unchanged. If for any reason narcosis should be prolonged after fifteen minutes, ether, nitrous oxide or ethylene may be administered, beginning about ten or twelve minutes after the commencement of the evipal, that there be no interruption. Pre-operative medication is not necessary or indicated. For any post-operative pain codeine or morphine may be administered.

At the Veterans' Administration Facility, Kecoughtan, Va., evipal has to a considerable extent replaced ether as a general anesthetic. In our eye service at this institution we have recently employed evipal soluble in nine cases. The age of these patients being in the forties and fifties, and their behavior being similar, it is not necessary to report each case in detail.

Evipal Soluble is supplied in ampules of .5, and 1 gm. of the powder inclusive with a 10 cc. ampule of distilled water. The solution is not stable and therefore it is necessary to mix the solution just before one uses it in order to have a stable solution.

There are two methods of figuring the dosage: One is based entirely on the patient's weight: A maximum of 6 cc. of the 10 per cent solution per 100 pounds of the patient's weight; the other is based on the patient's individual reactivity to the drug, i. e., the rapidity and intensity of effect of the drug that one notices upon the patient immediately as the injection is being made. The depth of the anesthesia produced is dependent upon amount of anesthetic introduced. In the majority of the cases it only required from 2 to 4 cc. to produce unconsciousness. The maximum dose to be given at any one instant must not be over 10 cc. The administration is started like any intravenous administration. The injection is very slowly introduced while the patient counts very slowly; this also aids in slower administration, besides giving an index as to the immediate effect. When the patient has reached the unconscious stage, one should delay for a period of thirty seconds in order to have a complete circulation of the blood and then continue the injection to the desired depth of the anesthesia.

What are the immediate effects of an intravenous injection of evipal? First, the patient very quickly falls asleep after only two or more cc. of the drug have been administered. There may be a very transitory twitching of the face and hands but this is about all of the stage of excitement that one sees and is immediately followed by complete muscular relaxation. The respiration is not materially affected; at first it is somewhat superficial and rapid, but soon becomes regular. The blood pressure level may fall from 15 to 20 mm. of Hg. but there is not a tremendous upset as one often finds in other anesthetics. Only in very rare cases do we find any change in the pulse rate; it may become a little fuller, but soon returns to the pre-operative normal of the patient. Thus we have a patient quickly anesthetized—with a minimum of discomfort to him and a minimum of danger as well.

This anesthetic has been used extensively in the last several years, upwards of two hundred and fifty thousand cases being on record in which it has been used. The mortality from its use is below the small percentage of unexplained anesthesia deaths of other anesthetics. However, there are a few contraindications in its use that would hold for most any other anesthetic, namely: Advanced cardiac, renal or liver disease, general sepsis, diabetic or arteriosclerotic gangrene, strangulated hernia, ileus, peritonitis, meningitis and carcinomatosis. It is not meant by the above that they are absolute contraindications, but an analysis of the record of the pa-

*Read before the Seaboard Medical Association of Virginia and North Carolina, at Virginia Beech, Va., December 8, 1937.

XUM

tients who have died falls in this general group. In other words, a poor pre-operative risk indicates a poor anesthetic risk as well. Caution and conservative doses—total, as well as fractional—are indicated in the presence of obesity, cachexia, dehydration, advanced hepatic disease, low blood pressure, cardiac or renal disease, sepsis senility, and alcoholism. I may add, further, that the drug is contraindicated in inflammatory processes of the throat in bronchiectasis and when respiratory obstruction exists. The administration of all barbiturates may produce spasm of the larynx and thereby cause mechanical obstruction *per se* at the site of the previously occluded larynx which otherwise would not cause any trouble at all. On only very rare occasions do we have any respiratory depression and this can be overcome by artificial respiration.

Today I shall only discuss one use of evipal anesthesia. I have listed its characteristics, its administration and dangers. Its uses in the field of medicine and surgery are legion, and I have been greatly impressed by its wide adaptability in all fields of medicine, surgery, obstetrics, gynecology, urinology, and orthopedics. However, my personal contact has only been in its application to ophthalmology. As a result of reviewing the literature in search for an anesthetic that would be quick in action, simple in administration and leave no after-effects, I find that evipal has no superior. In the last three months I have limited its use entirely to that of ophthalmology. My series of cases has each been enucleation of the eye—a procedure requiring a good anesthetic, and each of the series is classified under emergencies. A general anesthetic usually crowds the operative field; therefore the benefits from the operator's point of view in the use of evipal are many.

The series of cases, nine in all, are so alike typically that any one would serve the purpose as a whole. The average dose of evipal was six cc., and the shortest time before anesthesia ensued after the beginning of the anesthesia was before the patient could count nine in four second intervals and the longest was twenty. The average period of surgical anesthesia was twenty-one minutes. The patient when put to bed can be aroused and will respond to questions, but, if put in a dark room and not bothered, will usually sleep for about two hours. Our cases ranged sleeping from one hour and a half to two hours and twenty minutes. Perfect relaxation was obtained in all of the cases and the operation proceeded without the slightest difficulty.

I believe we have in evipal an anesthesia that will be a boon to ophthalmologists whose work is in many cases painfully tedious. I present this preliminary report to you to show one of the many special uses of the dose. I hope to prove to myself in the coming year a further extension of its use in the field of ENT. I am optimistic that its use can be so extended and will be the anesthesia of choice in this field also.

In concluding this short report, I believe we have an anesthetic here that can serve the specialist as well as the general man, for at times we all have painful procedures where the usual general anesthetic or even a local anesthetic would be impossible, and only an anesthetic of this nature would suit. Let us hope that many of us will try this in our respective fields and be as pleased as I am with the results.

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SCIATICA.*

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Sciatica is one of the disabling afflictions of the prime of life. Rarely does it affect a patient before he reaches his majority. It is as common in one sex as in the other. Domenico Cotugno,[1] an Italian, in his book "De Ischiade Nervosa," gave a complete clinical description of sciatica toward the middle of the eighteenth century. To this same man we owe the first accurate description of the cerebro-spinal fluid. Gussenbauer,[1] was the first to note the frequent association of sciatica with spinal deformity, although it remained for the Frenchman Brissaud,[1] to coin the term "sciatic scoliosis", to describe the spastic lateral curvature of the spine which often accompanies sciatica. Goldthwait,[1] of Boston, in

*Read before the Seaboard Medical Association of Virginia and North Carolina at Virginia Beach, Va., December 8, 1937.

1911, by his studies of the pathologic anatomy of the vertebral column opened up a new field for consideration of the etiology of sciatica from a mechanical point of view. The next important work on this interesting subject was that done by Danforth and Wilson,[2] in which they demonstrated anatomically the possibility of the lumbosacral region causing mechanical nerve irritation and hence sciatica. This work was substantiated clinically by Putti. In 1934, Ober[3] found that many patients with sciatica had an abduction contracture of the hip on the painful side and thought that possibly this mechanical derangement might be the cause of the pain in the thigh. Later, Freiberg[4] found that variations in the relationship of the sciatic nerve to the pyriformis muscle existed and that these variations probably caused sciatica in many instances by direct inflammation or mechanical irritation of the sciatic nerve.

Sciatica may be defined as pain radiating along the course of the sciatic nerve and its end branches. It is a symptom of trouble elsewhere and not a disease. The pain may be uniformly distributed but occasionally there are certain areas where it is more intense. The pain may begin abruptly or gradually. If the onset is abrupt the pain is usually sharp and shooting in character. If gradual, the onset is a dull ache, progressing into an intense pain radiating from the gluteal muscle down the posterior lateral aspect of the thigh to the calf muscle and occasionally into the foot. It may or may not be associated with low back pain.

The causes advanced for sciatica are as numerous as one finds for almost any other puzzling disease. One reason we have never been able to definitely localize the causative factor or factors is that sciatica, *per se*, does not cause death, so autopsy material for study has been very meagre. Furthermore, resection of the sciatic nerve *in vivo* along its course is impractical; therefore we have been called upon to theorize and to treat these patients by a trial and error method.

The following causes have been advanced for sciatica:

Foci of infection;
Pressure on the nerve within the pelvis;
Diabetes and chronic alcoholism—from a true neuritis;
Spinal cord tumors;
Relaxation of the plantar arches;
Subluxations of the sacro-iliac joint;
Unstable lumbosacral joint;
Anatomical changes in the relationship of the sciatic nerve to the muscles along its course.

Foci of infection are so well recognized by the profession as being the primary cause of inflammation in remote muscles, bursae, joints and even nerves that it is only necessary to remind you in passing to look for and eliminate them.

Pressure on the nerve from within the pelvis must be considered—from such causes as an inflammatory exudate, hardened impacted feces or a tumor mass. Careful bimanual examination in women or a rectal examination in men may easily eliminate these possibilities.

A true neuritis of the sciatic nerve as the result of diabetes mellitus or alcoholism is rare. The former may be ruled out by laboratory tests and the latter by the patient's history.

Spinal cord tumors may cause pain along the course of the sciatic nerve, but the sensory disturbances are soon followed by motor disturbances. Pain due to a spinal cord tumor is intensified by coughing, sneezing or straining. Included in the group of spinal cord tumors are herniations of the nucleus pulposus following rupture of the annulus fibrosis and hypertrophy of the ligamentum flavum. If in the course of the spinal fluid examination a block, partial or complete, is encountered or the spinal fluid protein is increased, tumor should be strongly suspected and lipiodal injection followed by roentgen ray examination should be done to localize the tumor mass. This examination is best done on a tilt top X-ray table with a "spot" film device, so that you may catch the lipiodal passing by the block if one is present.

While the organic lesions just described cause sciatica, they are present in the minority of patients suffering from sciatica in my opinion. Most of these cases are based on mechanical derangements and due in many instances to a decompensated muscular system.

One of these mechanical derangements is relaxation of the plantar arches, by which a pull is placed on the plantar and deep peroneal nerves which are the end branches of the sciatic nerve. By such a pull the strain may be transmitted to the sciatic nerve, or interpreted as being present in the thigh of the patient. I have seen a few dramatic cures by the simple expedient of placing an arch support in the shoe of a sufferer of sciatica.

XUM

Subluxation of the sacroiliac joint is one mechanical lesion that has been so well advertized by the profession to one another and to the lay public that it has been almost universally accepted as the cause of low back pain and sciatica by anyone who has any pain in this region. Actual subluxation of the sacroiliac joint is rare except in those cases following severe trauma to the pelvis. The sacroiliac joint is made for stability and not mobility. It has the strongest ligamentous support of any joint in the body and permits only a slight amount of rotary and gliding motion. Furthermore, the lumbosacral plexus of nerves from which the sciatic nerve arises is only contiguous and not directly associated with this joint. As added proof, tuberculosis and suppurative arthritis of this joint seldom ever cause sciatica. Except for ligamentous and muscular strains and inflammations I do not believe that this joint is often the origin of sciatica.

Let us consider why the anatomical set-up in the lumbosacral area may cause nerve disorders. Anatomic and roentgenographic examinations show that the lumbar invertebral foraminae are not all of the same size. The foramen between the fifth lumbar vertebra and the sacrum is the smallest in the group, that between the fourth and fifth lumbar is slightly larger, while those between the other lumbar vertebrae are still larger. In contrast to the smallest invertebral foramen between the fifth lumbar and the sacrum we find that the fifth lumbar nerve root is the largest of the group, the fourth nerve root is slightly smaller and the third nerve root likewise smaller still. Due to these changes, the fourth and fifth lumbar nerve roots are predisposed to injury by any change that may alter the size or shape of the foramen through which they pass. As these two nerves pass through the foraminae they are devoid of a protective arachnoid sheath and are surrounded by a rich plexus of veins. Therefore, any venous congestion that may arise in this area may cause pressure on these two nerve roots. These two nerve roots (fourth and fifth lumbar) are the largest branches to the lumbosacral plexus and form the greatest part of the sciatic nerve.

The lumbosacral joint is further predisposed to trauma and hence arthritic changes, as it bears the brunt of the superincumbent weight of the trunk and upper extremities. It is the meeting place of the movable spine with the relatively immovable pelvis. At this juncture we find various congenital abnormalities that may predispose to strains. Among these abnormalities are rudimentary articulations, and asymmetrical articulations, anterior and posterior displacements of the fifth lumbar vertebra on the sacrum, an increased lumbosacral angle, thinning of the intervertebral disc and unilateral or bilateral sacralization of the fifth lumbar vertebra. Any of these anomalies by deranging the smooth functioning of the joint may cause traumatic arthritis and hence a narrowing of the intervertebral foramen and pressure on the nerve roots. Also, any displacement of the fourth or fifth lumbar vertebra may cause a narrowing of the intervertebral foramina and hence pressure on the nerve roots. You may ask the question, "If these congenital anatomical features are the cause of pain in the back and legs, why don't they give rise to symptoms earlier in life?" The only explanation that I know of is that when we are young and active our muscles have a greater power of elasticity so that they function with the highest degree of efficiency, but as we become more sedentary in our habits these same muscles lose their elasticity (they become decompensated) and do not respond to sudden demands; therefore, instead of absorbing the shock and protecting these deranged joints, the force is directly applied to the joints and they degenerate, resulting in a reduction in the size and shape of the intervertebral foramina.

Another explanation for sciatica may be found in the muscles which are our first line of defense to trauma to the joints, as I have just pointed out. When we have pain, muscle spasm is Nature's method of protecting the injured part, and this is one of the objective signs that we find in an injured back or inflamed part. The muscles directly involved in the question of sciatica are the erector spinae and those connecting the pelvis with the hip and thigh. The gluteus maximus muscle has fibers of insertion or connection with the tensor femoris fascia, which in turn forms the mass overlying the sciatic nerve. If the spasm of these muscles continues for any length of time a contracture may ensue and this may be the cause of pain long after the original cause has subsided. Such may be the mechanism for the cause of sciatica in some instances for we know that stripping of the gluteus maximus muscle downward from its origin about the posterior superior spine of ilium, as described by Heyman,[5] has relieved some patients of their sciatica. Also severance of the fascia overlying the tensor

fascia femoris muscle, as described by Ober, relieves some of these patients. Both of these operations accomplish their purpose in all probability by releasing the bow string formation that these two structures form and thereby overcomes the abduction, flexion contracture of the hip joint, relieving the strain placed upon the lumbar spine and/or taking the direct pressure off of the sciatic nerve in the posterior thigh.

Another congenital muscular abnormality that deserves attention is the relationship of the pyriformis muscle to the sciatic nerve as brought out recently by Freiberg. The bifurcation of the sciatic nerve into the tibial and peroneal branches usually takes place in the upper portion of the popliteal space. However, in 10 to 15 per cent of the cadavers examined this branching takes place above the pyriformis muscle. When this branching takes place above the pyriformis muscle the peroneal nerve either goes over or through the muscle while the tibial nerve remains in its usual location under the muscle. As a result of such an anatomical peculiarity, any inflammation or contracture of the pyriformis muscle may be transmitted to the nerve and usually to the peroneal branch. We know that in almost every instance when the pain extends below the knee the distribution follows the course of the peroneal nerve. Freiberg states that Lasegue's sign —which is flexion of the thigh on the pelvis with the knee flexed and then extending the knee—is the best means of differentiating whether the pain reproduced is due to stretching of the pyriformis muscle or strain of the low back. This maneuver stretches the pyriformis muscle long before it stretches the nerve itself or the back muscles. I have personally seen two such cases operated on, and release of the impinged nerve from the muscle gave complete relief from the sciatica.

TREATMENT

Following an exhaustive examination and deduction as to the causes for sciatica in a particular patient, plan your form of therapy to relieve the pain at its origin. Except in certain instances, such as tuberculosis of the spine or severe mechanical abnormalities as a spondylolisthesis which require a spine fusion for cure, the treatment should be conservative in the early stages.

Conservative treatment should consist of rest on a firm bed, local heat, massage, and, in some instances, Buck's extension, using only enough weight to relieve the muscle spasm present. As to the form of local heat applied, I believe the deep or penetrating heat—as that produced by diathermy or the short wave apparatuses—to be the best and will often relieve an acute attack in one to three applications. The procedures just mentioned are all directed towards relieving local inflammation and in many instances are all that are necessary. In other instances the patient will require support for the low back and pelvis. Usually a belt will suffice in men, but in women a corset is much more comfortable.

The use of epidural injection—employing 1 per cent novocaine and saline to break up meningeal adhesions by the pressure of the solutions—has been advocated. Many men report some excellent results but so far I have never had the good fortune to relieve a patient by this method, although I have tried it several times.

Stretching of the sciatic nerve on the involved side is a blind procedure and is to be condemned. If one believes that adhesions are present about the nerve they should be released by open operations. The part of this procedure that does the most good in my opinion is the rest in a plaster cast that follows the stretching.

The only place that drugs have is to ease the acute pain while attempting to localize the origin of the trouble. Among these drugs, the salicylates are the best to employ. Morphine should not be used because of the likelihood of making the individual an habitué. I have recently had a patient who stated that she became a morphine habitué because of intractable sciatic pain.

Intraspinal injections of alcohol to relieve sciatic pain are too dangerous to employ in such an affliction. This procedure is permissible to relieve the intractable pain due to malignancy where, if a paralysis results, no great harm has been done.

If the patient fails to respond to conservative treatment that has been properly carried out, you are then justified in attempting one of the many operative procedures. Among these procedures is the one devised by Ober and this has been highly successful in a selected group of patients. Those patients who have an abduction—flexion contracture of the affected hip—are the ones in whom you may expect the greatest relief. The test for this deformity consists in attempting to abduct the extended abducted thigh with the knee at a right angle, the patient lying on his side. The test is positive if the

XUM

knee cannot be brought to the table but remains in an abducted position, and a tight iliotibial band may be palpated. Division of this tensor femoris fascia is done just above the trochanter and usually under local anesthesia. The patient is required to stay in the hospital only two to three days and the procedure offers such remarkable results that I see no reason to unduly prolong the conservative treatment in this group of patients.

As this procedure of Ober's is so simple and offers such a high percentage of relief from sciatica, I believe it is much better to try it before an exploration of the sciatic nerve and pyriformis muscle is made, as suggested by Freiberg. However, I do believe that the relationship between the sciatic nerve and the pyriformis muscle is responsible for a great many of the sciaticas that occur without an associated low back pain.

One argument against the Ober operation is that we are operating on a symptom and not the cause of the pain. This may be true, but we are all interested in giving our patients relief, and this procedure has been able to do it in many instances. True, we do not know yet just how long these patients will be relieved of their pain, but if they are relieved for only a year the procedure is worth while to those patients.

If all of these procedures fail and we have definite mechanical defects in the low back, it is then time to consider a spine fusion, operative enlargement of the intervertebral foramina if it is narrowed by an arthritic spur, or removal of a herniated nucleus pulposus.

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A CASE OF CONGENITAL MEGACOLON TREATED BY DIATHERMY.*

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There is considerable evidence in favor of the opinion that congenital idiopathic megacolon is in many instances caused by an imbalance in the autonomic nervous mechanism of the colon and rectum. The colon and rectum receive their nerve supply from both the sympathetic and parasympathetic divisions of the autonomic nervous system. The sympathetic nerves inhibit the muscle of the bowel and are motor to the internal anal sphincter, while the parasympathetic nerves produce the opposite effect. A considerable number of cases of congenital megacolon have been greatly benefited and some apparently cured by the resection of the sympathetic nerve supply of the lower part of the bowel.[1, 2, 3, 4, 5] It would nevertheless be desirable to avoid surgery if relief could be obtained by other methods.

Harmeier and Rowntree[6] showed in 1930 that the application of diathermy to the cervico-thoracic region of a patient afflicted with chronic infectious arthritis produced a temporary rise in skin temperature and an increased mobility of the upper extremities. This effect resembled that previously observed after sympathectomy in cases of arthritis. Diathermy applied locally to the affected parts caused no relief. Since the sympathetic nerves supplying the lower bowel take origin in ganglia which are located in the abdominal area, anterior and anterolateral to the spinal column, it might be thought that the application of diathermy to the abdominal region would cause changes in the action of the bowel musculature resembling those observed after sympathectomy. Amberg,[7] in 1930, reported a case of congenital megacolon which was successfully treated by diathermy, one electrode being applied over the lower dorsal and lumbar vertebrae and the other over the lower part of the abdomen. The beneficial effects were striking, and at the time of reporting had persisted for three months after the

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treatment was stopped. In the case reported at this time, sympathectomy was first considered, but it was decided to determine the effects of diathermy and to resort to surgery later if necessary.

CASE REPORT

The patient, a boy six years old, was admitted to the Sheltering Arms Hospital on July 2, 1936. His chief complaint was persistent constipation which has existed since birth. It was stated by his mother that he had rarely had a spontaneous bowel evacuation, and then only with great effort and straining after a week or more had elapsed without enemas. After some time without any bowel movements he had often become distended with gas and feces, and had suffered much pain. He had usually had a poor appetite and had always been under-weight. He had always tired easily. At the time of admission he had not had an evacuation for three days. Examination revealed the following significant findings: The patient was a rather under-nourished looking boy with poor muscular development. The abdomen was much distended with gas and hard fecal masses, and there was active visible peristalsis with borborygmus. The ribs flared outward, and the heart was pushed upward by the abdominal contents. A soft systolic murmur was heard in the third intercostal space at the point of maximal cardiac impulse. It was impossible to outline the lung bases by percussion. The circumference of the abdomen at the level of the navel was twenty-four and one-half inches, and at the costal margin was twenty-eight inches. The weight three days after admission was forty-three pounds. A roentgenogram of the chest showed considerable elevation of the diaphragm. Roentgenographic examination of the gastrointestinal tract revealed no abnormalities in the stomach and small intestine, but showed marked dilatation of the descending colon, sigmoid colon, and rectum. On the basis of the history and physical examination a diagnosis of congenital megacolon was made.

Treatment was begun by attempting to reduce the distension of the abdomen. A low residue diet was prescribed. Small saturated magnesium sulphate enemas, and later normal saline enemas, were used with fairly good results. There was complete disappearance of the distension by the third day, but one large fecal mass persisted in the lower right abdominal quadrant. On the fourteenth day (7-16-36) short-wave diathermy treatments were started. These treatments were given once daily for twelve days, one electrode being placed over the lower thoracic and lumbar vertebrae and the other over the lower abdominal area. Each treatment lasted twenty minutes, and a current of 1,000 milliamperes was used. During the first five days of diathermy treatment the patient had a daily spontaneous bowel movement. Daily enemas were necessary during the next five days, possibly because of the persistence of the fecal mass in the lower right quadrant. It was later discovered that the treatments were improperly applied on these five days, since, due to an error by the intern, the electrodes were so applied as to produce principally skin heat with very little deep heating effect. On the tenth day of diathermy treatment three ounces of a 50 per cent solution of hydrogen peroxide were injected into the rectum for the purpose of promoting disintegration of the fecal impaction. Though most of this was expelled promptly, a normal saline enema given an hour later was very effective. There followed a period of three days during which the patient suffered from epigastric pain of considerable severity, and lost his appetite almost entirely. There was no muscular rigidity. The temperature rose to a maximum of 102.5 (by mouth), returning to normal when the pain stopped. Daily spontaneous bowel evacuations began on the day after the hydrogen peroxide enema was given and continued for twenty-five days, without enemas or laxatives, until discharge from the hospital (8-22-36). Shortly before leaving the hospital the patient's weight was forty-five and one-half pounds, a gain of two and one-fourth pounds since admission; the circumference of the abdomen at the navel was twenty-one and one-fourth inches and at the costal margin twenty-four and one-fourth inches, a reduction of three and one-fourth inches and three and three-fourths inches respectively. He had improved considerably in appearance, there being better color and more subcutaneous fat on the face and extremities. During the last twenty-one days in the hospital he was given a soft diet.

A letter from the patient's mother three weeks after leaving the hospital stated that he was again requiring daily enemas, but that he was in excellent health otherwise, had no abdominal distension, and weighed forty-eight and one-half pounds.

SUMMARY

A case of congenital megacolon is reported in which treatment by diathermy was followed by re-

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lief of symptoms which continued at least twenty-five days.

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THE USE OF TURNBUCKLES IN TREATMENT OF FRACTURES OF THE PELVIS. A PRELIMINARY STUDY OF FOUR CASES.

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In the fall of 1936, we received a reprint on the treatment of fractures of the acetabulum by the use of turnbuckles and I regret to say that this was lost and I can not remember who did the work or the technique he used, but it impressed me as a very good conservative way to treat these cases. Since that time I have used it in three of these cases and in one case of wide separation of the symphysis.

In a wide search of the literature I have been unable to find this article or any other on this method; consequently, I am unable to give credit for the origin of this method to the originator. Its simplicity and effectiveness impressed us in the few cases in which it was used.

Nearly all central fractures of the acetabulum are caused by direct trauma over the greater trochanter, forcing the head of the femur inward. The majority of these cases are accompanied by other fracture of the pelvis, usually the rami of the pubes, and quite often with some dislocation or fracture of the sacro-iliac joint.

The treatment should be directed so as to prevent any deformity and give good function. These fractures of the acetabulum tend to give a traumatic arthritis with limitation of motion and a painful joint.

TECHNIQUE

After the diagnosis has been made and complications either ruled out or treated, the patient is ready for reduction. This should be done as soon as it is possible, usually after two or three days when all signs of shock have passed. Without anesthesia, a plaster of Paris cast is applied on both legs, from the toes to the groin, with the bony points well padded and with a basswood splint incorporated on the medial aspect to distribute the pressure. Then a short turnbuckle which is closed is incorporated as near the perineum as possible (this turnbuckle is about four inches long) and a long turnbuckle about sixteen or eighteen inches long is applied open about the middle of the leg. After the plaster has set, the small turnbuckle is gradually opened and the long one closed, thus acting as a fulcrum forcing the head of the femur out of the acetabular cavity. This technique is reversed in separation of the symphysis and it is forced together. I recall from the unknown author of the reprint all cases reported were fractures of the acetabulum, but by reversing the technique the turnbuckle works even better on separation of the symphysis.

A SHORT REPORT OF CASES

J.H.B. was admitted to hospital on January 5, 1937, following an automobile accident four hours before in which he was thrown from the car and got severe brush burns of the face and arms and hurt his hip.

Examination on admission showed a boy, age 16, in pain and some shock. Blood pressure was 75/40.

There were several small abrasions over the face and arms; also there was marked tenderness over the symphysis pubis with a defect at this point. Marked pain was caused by moving and by compression of the crest of the ilia. Abdomen flat and soft. Catheterized specimen showed gross blood.

X-rays revealed marked separation of the symphysis pubis and a fracture of the rami of the right pubic bone.

He was treated for shock and his bladder was observed for eight days. Then turnbuckles and cast were applied. Four days later, X-rays revealed a reduction of the symphysis and a bilateral spica was applied. He was allowed to go home in four days and kept in this cast for eight weeks, then allowed up on crutches. Four months later, X-rays revealed healed fracture of the pubic bone, symphysis still in contact, and clinically he was completely cured without any discomfort.

L.M. was admitted to hospital on June 15, following an automobile accident in which he was thrown out and landed on his right hip. Examination and X-rays revealed a central fracture of the right acetabulum and fracture of the rami of the right pubis with a partial dislocation of the right sacro-iliac joint. No complications were found, so cast and turnbuckles were applied after two days. Reduction completed in four days and spica applied. Cast removed in eight weeks and the patient allowed on crutches. Six weeks later, patient was walking without a limp and clinically was not restricted in his motions and not complaining of pain.

A.G., twenty-one, male, fell from a tree, hurting his right hip. Examination and X-rays revealed central fracture of the right acetabulum and fracture of the rami of the pubic bone. After three days, fracture reduced with turnbuckles and spica applied, the patient being allowed to go home. Cast was removed by family physician who stated patient had good motion, but was complaining of some discomfort four weeks after removal of cast.

E.G., colored female, forty-five, admitted to hospital following an automobile accident. Examination and X-rays revealed a central fracture of the right acetabulum with fracture of the rami of the pubis on the same side. No complications appeared and two days later a cast and turnbuckles were applied. Six days later reduction was complete and spica was applied. Cast removed in eight weeks and the patient was allowed up on crutches. Time is too short to judge the final results in this case.

CONCLUSIONS

Few conclusions, if any, can be drawn from so few cases.

1. All four had multiple fractures with displacement.

2. Position of fragments was improved in all cases.

3. In three of the four cases, which have been followed over six months, the results—so far as function is concerned—have been excellent, but one of the cases is still having discomfort about the hip. The fourth case has not been out of her cast long enough to determine the results.

4. The stay in the hospital in uncomplicated cases averaged about eight days.

307 Professional Building.

OVER-ACTIVITY AS A POTENT FACTOR IN THE ETIOLOGY OF SOME NERVOUS DISEASES.

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Having had an opportunity, during a number of years, to observe individuals working in lead, a problem presented itself for solution. Some workers showed left-handed wrist-drop, others right-handed; another had a double wrist-drop; another had paresis of the right leg. In all these cases there was an absolutely identical etiologic factor, and still the functional disability did not manifest itself in the same portions of the body. In several professional pianists I have observed attacks of cramp-like sensations in the wrist, and in others in some of the fingers. The occupation and the continuous efforts being the same, the seats of the disorder are not identical.

In a large group of tabetic patients, I had three cases of the cervical type in which ataxia began in

the upper extremities. Why, therefore, is the functional disorder more marked in the arms than in the lower extremities, when the specific infection is of a general character? In some of my tabetic patients the eye symptoms were conspicuous from the beginning. In others the leg symptoms were the first to appear and the eye disturbances appeared quite late in the course of the tabes. In some of my cases the involvement of the sphincters was a late manifestation; in others a very early one. These differences in the onset and localization of functional disability in various persons suffering from tabes dorsalis are striking and require an etiologic explanation, especially in view of the same syphilitic toxic agent in all cases of tabes.

Why is it that paresis is most frequent in persons who exercise their intellectual more than their physical functions? I had four patients with juvenile paresis in whom the symptoms made their appearance with their entrance into college, otherwise speaking, at the time of greater demands on intellectual efforts. What explanation can one find with regard to such an onset?

In a group of cases of progressive muscular atrophy of spinal origin, the affection began in the upper extremities in some patients, in the lower in others; in one limb in some cases and simultaneously in two symmetrical limbs in others.

The largest majority of my poliomyelitic cases were in children, in whom the affection was confined to the lower part of the body. In five adults the functional disability was observed in the one or both upper extremities. Three of these individuals were tailors and two were bricklayers. What is the reason of the difference in the localization of the pathologic process in the children and adults?

In three cases of pernicious anemia in men there were evidences of a degenerative state of the spinal cord. In one case of the same affection in a middle-aged woman the central nervous system was not in the least involved. If a toxin is supposed to be the cause of the pathologic process of the cord, wherein lies the difference in the cases of the men and of the women?

Weigert and Roux long ago expressed the view "that normally there is a state of equilibrium among all cells of the body, and their interrelation is such that when one cell becomes diseased or otherwise disturbed, the energy of the cells which lie close to it develop an increased energy, namely, prolifieration, and, *per se*, repress the affected cell still further. Weigert also calls attention to the fact that the process of functioning of a tissue is accomplished by its destruction, which in normal conditions is rapidly compensated by a proper supply of nutrition. But if the latter is not forthcoming, the above-mentioned equilibrium is disturbed and degeneration takes place. Edinger went further and said that if, instead of—or with—a defective nutritive supply there is an excessive functionating of a cell, the increased growth of the neighboring cells will lead to a degeneration of that over-active cell, which naturally is then less resistant. This is particularly seen when both elements are at work, namely, over-activity and nutritional deficiency.

Edinger succeeded in demonstrating by some experiments the rationality of his contention. It is therefore evident that over-activity or superfunction is a powerful predisposing factor in producing diseased conditions in an individual whose organism is potentially under the influence of some toxic or otherwise abnormal element. Since function is apparently a potent agent in creating a syndrome of abnormal phenomena, it will be of interest to analyze closely a few of the diseased processes of the nervous system from that particular standpoint

Occupational Neuroses.— There were thirty-seven patients distributed as follows: twelve stenographers, five newspaper reporters, three violinists, six pianists and eleven shoemakers. For many months without interruption they strenuously carried on their work. Moreover, they all appeared to be underfed. In the stenographers and reporters the localization and character of the disorder were not identical. Some of them had the paralytic form, namely, a sudden sensation of fatigue and numbness in the hand while writing. In others there was an actual cramp, namely, a sudden extension of the index finger and flexion of the thumb. In others a cramp appeared in the wrist, but not in the fingers. One of the three violinists developed a cramp in the right hand and the other two in the left hand. Of the six pianists, in two a sudden paretic condition would set in in the left wrist; the other four had a similar condition in the fingers of the right hand. Of the eleven shoemakers, six presented attacks of cramps in the biceps muscles of one or the other arm: the remaining five individuals had attacks

of numbness in the thenar muscles of one or the other hand.

A close analysis of these thirty-seven cases convinced me that the occurrence of the neurosis in one particular segment or portion of a limb was due to a predominance of effort practiced by the affected region. Thus, the violinists who had attacks of cramps in the left hand happened to be left-handed. The two pianists with the paretic attacks in the wrist were trained by teachers whose method consisted of holding the wrist stiff during the exercises.

Chronic Lead Intoxication.—The next group of cases in my series comprises sixteen cases. Five of them had double wrist-drop. Two men had wrist-drop only on the right side; three men had wrist-drop only on the left side; three men had wrist-drop on the right side. Two men, typesetters in a printing house, presented a paretic condition of the three first fingers on the right side. Three men were house painters who used a small size brush for their work. They presented wrist-drop on the right side. One individual was supervisor in a white lead factory for ten years. He had to spend the entire day in the lead atmosphere. He developed a paresis of the right leg with a markedly diminished knee-jerk and Achilles tendon reflex. All the patients of this group had general evidences of plumbism, namely, blue line, obstinate constipation, albuminuria, headache. An inquiry into the character of the work of the sixteen patients revealed the following: The individuals with double wrist-drop worked for a number of years in white lead factories where both arms were used with equal effort. The men with unilateral wrist-drop worked in lead colors, and their work consisted of mixing the dyes, which they did with one or the other arm. Unilateral extensor palsies in plumbism are not a frequent occurrence. Nevertheless, in the few cases which I studied from the standpoint of exhaustion, I found that the nature of employment coincided with unilaterality of paralysis. The two type-setters, all right-handed men, used in their work chiefly the thumb and the first two fingers continuously. Although the general symptoms of saturnism were present, nevertheless the local symptoms were conspicuous in those fingers which are most persistently in use and therefore most readily fatigued. The three house painters, being compelled to extend the wrist thousands of times in succession, naturally developed a paralysis of the extensor muscles of the wrist. All being right-handed, they presented a right-sided wrist-drop. The supervisor of the lead factory who developed a paresis of the right leg had to watch over groups of men. In making his rounds he would stop at a certain group and remain in one sitting position for an hour at a time. While sitting he would place the right ankle on the left thigh and keep up that position during the entire hour. He repeated this position at each group of men whom he was obliged to inspect. This attitude was considered his habit, and he was never seen at the factory in another position. His wife corroborated this statement and said that that was also his sitting position in his home.

Poliomyelitis.—The poliomyelitic cases comprise a group of thirty, among whom there were five adults and the others were children. Of the former three were tailors and two bricklayers. One of the tailors had the right upper and lower limbs involved. Another tailor had both upper extremities at first stricken, but later the left arm recovered. The third had the right arm and right side of the face paralyzed. Evidently here the cervical cord as well as the medulla was affected. The two bricklayers had both their right arms and both lower extremities involved. The identity of the condition in both patients was striking. It is evident that the five adults whose chief exertion was manifest in the upper limbs had the cervical portion of the cord involved, as the ganglionic cells of this part of the central nervous system supplied nerve fibers to the brachial plexus, hence to the upper extremities. Of the twenty-five children, ten had, in addition to one or both lower limbs, also one or both arms involved. Inquiry revealed that they belonged to very poor families, and being from eight to twelve years of age, they had to do considerable amount of manual work at home such as scrubbing, cleaning, etc. Thus the involvement of the upper limbs can be readily conceived. On another page the usual occurrence of the poliomyelitic process in the lower extremities in children was mentioned. The reason of it lies in the great activity of the limbs, which are in a state of development in young children.

Progressive Muscular Atrophy.—In a group of

fifteen cases of progressive muscular atrophy of spinal origin, the affection began in the lower extremities in one policeman and in one watchman; in two Italian street cleaners in one upper limb. The remaining eleven cases showed an onset in both hands. The latter patients were all either laborers or tradesmen. The occurrence of the disorder in the lower limbs of the policeman and watchman speaks strongly in favor of the view in my thesis.

Pernicious Anemia.—There were four cases of pernicious anemia in my series. In the men, three in number, evidences of degeneration of the spinal cord were present. They presented symptoms of ataxic paraplegia, with diminished or absent knee-jerks. Two of them were laborers and one a collector for an installment house. The fourth patient, a woman, housewife, presented no disturbances of locomotion and the knee-jerks were normal. Here again we have a corresponding link between the degree of over-activity of certain portions of the body and an organic lesion of that part of the nervous system which controls the affected portions.

Paresis.—Out of thirty-nine paretics twenty belonged to the class of individuals who lead a purely intellectual life. Teachers presented the majority. In them the mental symptoms were the most conspicuous. With the exception of the eyes, other physical symptoms were very slight, namely, tremor of hands, slightly increased knee-jerks and no toe phenomenon. Nineteen patients were tradesmen in whom the physical as well as mental symptoms were equally developed. They presented a condition called "tabo-paresis," namely, symptoms of tabes, together with mental disturbances found in general paralysis of the insane. If we consider the combination of physical and mental efforts displayed in the occupation of tradesmen, we must admit that the above observation as to the morbid manifestations in these individuals is of no little interest and is more than a mere coincidence. Four patients of the entire group had juvenile paresis. Their ages ranged from sixteen to twenty. The mental manifestations made their appearance at the time of their entrance to college. It is a period of intellectual life when application and mental efforts are tested at their utmost. It is also highly interesting to notice the variability of the eye conditions in the paretics. Among the tradesmen there were two jewelers and three bookkeepers. It is remarkable that in every one of these men optic atrophy and Argyll-Robertson pupils were very striking and developed early in the disease. In the persons who led a purely intellectual life there were twelve teachers. All presented pupillary symptoms and some also palsies of the third nerve. Five cashiers of banks showed besides third nerve palsies also evidences of eye-ground changes. The inference is consequently the same as in the cases of tradesmen from the standpoint of the principle of "exhaustion."

Tabes.—In considering the group of cases of tabes, forty-seven in number, the inquiry was made as to the character of occupation and the degree of exertion of the tabetics. The largest majority (thirty-nine) were individuals who did a great deal of standing or walking and carrying heavy weights. In this group there were letter carriers, policemen, messengers, icemen, laborers and motormen of street cars. The lower extremities alone were at first involved and the tabetic process began with disturbances of locomotion. Out of the remaining eight patients, three presented at first ataxia of the upper extremities and diminution of knee-jerks, so that the diagnosis of cerebellar disease was originally entertained. Two of these patients were stenographers and the third, a woman, worked at a laundry. The stenographers' duties were particularly laborious and their hours were very long. They eventually developed leg symptoms.

In connection with the subject of tabetic ataxia in the extremities, it is well to recall what was said on the introductory pages with regard to the sensory neurons. The continuous and uninterrupted flow of impressions received by our peripheral sensory apparatus is the most important factor in all muscular contractions and in the display of our joints. Should the organism be invaded by a poison, such as syphilitic in tabes, the over-functioning tissues or organs naturally will suffer first, hence the disorder of locomotion and coordination, which so much depend on the function of the sensory neurons.

Proceeding further with the analysis of tabetic manifestations with reference to the principle of "exhaustion," the following is observed: Three patients who led a sedentary life worked in poorly lighted rooms, two of them, clerks in the same establishment, were assigned offices with windows opening in a very narrow and small yard, so that often they had to use gas light. The third had to

use gas light all day. He was cashier in a small store and his desk was in the back of the store. All three when first seen presented optic atrophy without ataxia, but with much diminished knee-jerks. Two patients were electricians working in very brightly illuminated rooms and glaring light for ten hours daily. They also presented optic atrophy as an early tabetic manifestation. At the International Congress of Medicine held in 1923 the effect of very bright light was considered as injurious to the eye, owing probably to the presence of ultra-violet rays. The early occurrence of optic atrophy in the two electricians is undoubtedly due to this latter circumstance.

Thirty-two patients of my entire tabetic series presented ptosis in one or in both eyes and Argyll-Robertson pupil in one or both eyes. If we consider the extraordinary activity and the incessant display of the light reflex from the moment we awake in the morning until we close our eyes for sleep—if, moreover, we consider the activity of the levator palpebrae which keeps the eye open—we cannot be surprised at the fatigue of these over-active functions in a body which is under the chronic influence of syphilitic toxemia, and therefore the early and frequent occurrence of Argyll-Robertson pupil and ptosis in cases of tabes are to be expected.

CONCLUSIONS

The present study, while it does not explain every morbid manifestation, nevertheless is sufficiently suggestive as to the effect of over-function of organs on their early morbidity. It teaches that a body which is potentially diseased, or which is under the influence of a disordered metabolism through the presence of a toxic material, will develop morbid phenomena primarily in parts functionally exhausted. The "exhaustion view," as we have seen, is demonstrated quite satisfactorily in syphilitic affections of the nervous system, in alcoholism, in plumbism, in acute infectious diseases such as anterior poliomyelitis, in profound anemias and in other conditions accompanied by profound metabolic disturbances of any nature. It explains the constant and almost uniform and earliest involvement of those portions of the body that are kept in a state of fatigue.

This observation points to a very important feature in the domain of prophylaxy. Bearing in mind the pathologic potentiality in cases of chronic toxemias, early treatment of the latter, combined with removal from the usual occupation at the earliest possible moment, is apt to prevent localized morbid manifestations such as we have seen above with regard to locomotion and eye manifestations in tabes, or localized palsies in intoxications, for example. A potential syphilitic individual will thus be able to avert for a long period of time the early on-coming of morbid manifestations of syphilitic or parasyphilitic character in the domain of the nervous system. The principle of prevention is here strikingly evident. The recognition and adoption of the view of "exhaustion" in affections of functional and organic nervous disease appears to be far reaching.

1900 Locust Street.

Correspondence

Real Service.

The following is taken from a letter of May 9, written by Miss Margaret Pritchard, R. N., with the E. L. Graham Hospital at Kwangju (Koshu), Korea, sent to the Southern Presbyterian Mission at Nashville, Tenn. As it tells of the work of two Virginians—Drs. L. C. Brand and James McLean Rogers—we feel it will be of interest to a number of our readers and will be but a small tribute to their service.

On the 19th of January Dr. Brand was taken very ill with a severe form of influenza. There followed a long illness resulting in his departure from this life, March 1, at 8 a. m. We all thought a week before he left us that he would be back at his work, which he loved so much, in a few weeks. He has left such a big vacancy that it is staggering to think about. He was a loving, thoughtful, unselfish physician and friend to Koreans and missionaries alike. The statement made at his funeral by the Korean pastor that every Korean in this section of Korea was poorer because of Dr. Brand's home-going is certainly true. The splendid medical plant and native staff that he leaves is certainly a fitting memorial to his almost eight years of fruitful service in Kwangju. The morning of his departure I told the staff that I knew that he would want the work to go on as usual. They courageously faced

the patients and the clinic was closed only during the funeral. If you could know how emotional these people are at such a time and how much they express their emotions you would appreciate their response. We can only say God's will be done and thank Him for the privilege that has been ours of being associated with such a Christlike doctor in the medical service here for almost eight years and strive to do our part in continuing to carry on the work. The day of Dr. Brand's funeral some Korean friends started a memorial fund with the hope that it will be added to, sufficiently to eventually erect a memorial that will be fitting.

Just when I was feeling the added burden of the work most after his going, Dr. Rogers volunteered to leave his busy hospital two days a week and give that time to this hospital until his furlough which starts in June. After that Dr. Preston who will have charge of the Soonchun Hospital in Dr. Rogers' absence will come one day a week. My first reaction was that God certainly provides for His work, but it is being done at a big price just now as far as our medical work is concerned. Dr. Rogers leaves a hospital with over 100 in-patients to come to help us. He arises at 4:30 the days he comes here and gets home at 10:30 p. m., makes rounds in his own hospital after that. By the time he gets to bed it is past mid-night and he has spent over nine hours of the day on a third-class coach with straight wooden-back seats. He does it joyously and gladly but we all know he cannot do that indefinitely. Dr. Boggs is the other doctor in our Mission and he is carrying the Chunju and the Kunsan Hospitals. Please pray that we may soon have help in this critical situation in which our medical work finds itself.

Dr. Brand leaves a very finely trained and capable native doctor in Dr. C. E. Rowe. Dr. Rowe is a splendid surgeon and a good medical man. We are very proud of him and most thankful for him. We feel that he has been raised up for just this time. The work has gone on without any slump. In fact we have had an unusually busy April. The second t. b. ward which was the gift of a missionary has been opened and has only one empty bed today. We have eleven cases in this department now and room for only two more.

I have just finished my annual report for the Mission and the past year's statistics are interesting to me, hope they will be to you. We have had 1,154 in-patients with a total of 9,728 in-patient days. Total operations 696. The clinic had 4,539 New patients with 8,010 return visits. There are enrolled in the free baby clinic 136 babies. Total receipts Yen 25,320.65. We received from the Mission Yen 1,320 and Dr. Brand's and my services. The medical work out here is most encouraging. The most discouraging feature being our lack of medical missionaries. We are rejoicing over the long-prayed-for nurse for Kunsan, Miss Elizabeth Woods, whose parents, three brothers, and one sister are missionaries to China. I was especially glad to welcome her since I was the only full-time nurse in our Korea Mission this year until she arrived, Misses Kestler and Hewson being on furlough.

I cannot close without telling you about our hospital baby. The wife of our hospital outside man died from tuberculosis leaving a tiny baby boy with no one to feed him or care for him but an old grandmother. Mr. Avison our Y.M.C.A. Secretary, gave the father a fine goat that furnishes food for the little fellow, our hospital is his home and our nurses take care of him. He is growing nicely and is a source of pleasure to all of us.

Why Not Remember Them?

Richmond, Va.
June 7, 1938.

TO THE EDITOR:

A few years ago I wrote a paper on—"Surgeons In The Confederacy", for the Richmond Chapter, United Daughters of the Confederacy.

When one does research on a particular subject one is more apt to take notice of things pertaining to that subject. Often in going through the cemeteries I notice the monuments of the soldiers buried there. When I see some so badly neglected with such inscription as—"Gone But Not Forgotten"—with weeds as high as your head and one has to push them aside to be able to get the inscription on the monument, it does make one stop and think. The men who saw the crimson blood shed for their Country's call—wounded and killed—that their graves should be so sadly neglected.

I have in mind one special surgeon—Hollywood Cemetery—JAMES B. SOUTHALL, SURGEON, C. S. A., DIED DECEMBER 10, 1865. The Virginia State Library shows that one James Southall enlisted from Charles City county, Va., 1864, and surrendered at Appomattox Court House.

Shall it be in vain that these men served their

country? Do you think there could be a Surgeon's Society formed to look into the neglected graves of the men who relieved the suffering of the men who fought so bravely for the protection of their homes, '61-'65? Perhaps an article written in some magazine would remind relatives of the neglect of these noble men—"Gone But Not Forgotten".

JULIA E. SAUNDERS
(MRS. WALLACE C. SAUNDERS).

Miscellaneous

Assembly of Laboratory Directors and Serologists.

The intensive campaign to stop the spread of syphilis now being waged throughout the country makes it imperative that only those serologic tests of proved efficiency be made available to private physicians and health officers. Diagnosis of syphilis must be prompt and accurate. The serologic blood test, becoming positive within two or three weeks after the onset of primary syphilis and remaining positive in the vast majority of untreated patients throughout the entire course of the disease, is the most important evidence of the existence of syphilis.

The Amerian Society of Clinical Pathologists in cooperation with the U. S. Public Health Service realized the need for reliable serodiagnostic tests several years ago. The work of the Committee on Evaluation of Serodiagnostic Tests for Syphilis is sufficiently well known to require no comment. It is the opinion of this Committee that its studies of the efficiency of the performance of serologic tests have progressed to a point where material gains would be made by a thorough discussion on common ground in which all those interested in the control of syphilis through laboratory methods may participate.

Plans are being developed for an assembly of laboratory workers from the entire country. All such workers both from private, hospital and public health laboratories, as well as physicians and health officers interested in the control of syphilis are invited to attend.

The proposed meeting, under the auspices of the Committee on Evaluation of Serodiagnostic Tests for Syphilis, with Surgeon General Thomas Parran, Chairman, is scheduled for October 21 and 22, 1938, at Hot Springs National Park, Arkansas.

The aims and purposes of the assembly will be to consider means and methods to improve and to make more generally available the serologic tests, which are so important in syphilis control work. Tentative arrangements call for the presentation of the program in four sections.

The first section will consider the need for abherence to conventional technic in the routine performance of reliable serodiagnostic tests. This subject will be considered in papers by Drs. Harry Eagle, William A. Hinton, Reuben Kahn, Benjamin Kline, and John H. Kolmer, with special reference to the tests which each of these workers has described.

Need for training of laboratory personnel will be the subject of the second section. The qualifications and training for both laboratory directors and technicians will be presented in separate papers.

The third section will discuss the prosecution of the studies to evaluate the performance of serologic tests within the States. The efficiency of branch State laboratories and of municipal, hospital and private laboratories cannot be studied on a national basis. The subject is much too large. Should this be made a function of the State or large municipal department of health? Actual experience with such studies in the States of Maryland and New Jersey and in the City of Cleveland will be described.

The fourth section will consider the desirability of licensing or approving for the performance of serodiagnostic tests for syphilis, laboratories within the States by the respective State departments of health. This discussion will be conducted from the standpoint of the private laboratory director by Dr. Frederick H. Lamb of Davenport, Iowa. The health officer's side will be presented by Dr. A. Wadsworth, State Department of Health, Albany, New York.

A separate committee will draft recommendations for each of the four sections for presentation to the assembly The respective chairmen of these four section meetings will be Drs. Walter M. Simpson, Dayton, Ohio, Arthur H. Sanford, Rochester, Minnesota, F. E. Senear, Chicago, Illinois, and H. H. Hazen, Washington, D. C. General discussion will follow the presentation of each set of recommendations.

An additional feature of the meeting will be an actual demonstration of the performance of the Eagle, Hinto, Kahn, Kline, and Kolmer tests by the originators of these procedures.

It is to be hoped that the attendance at this as-

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sembly will be large. Out of the meeting should come a crystallization of opinion with regard to the important problems which will be considered. Those interested in obtaining further information should write to the Surgeon General, U. S. Public Health Service, Washington, D. C.

Public Health Statistics

I. C. RIGGIN, M. D.,
State Health Commissioner of Virginia.

The report of the State Health Department's bureau of communicable diseases, as compiled for the month of May, shows the following cases compared with the same month in 1937.

	1938	*1937*
Typhoid and Paratyphoid	14	34
Diphtheria	42	39
Scarlet Fever	78	62
Measles	1,609	2,560
Meningitis	12	42
Poliomyelitis	1	3
Rocky Mountain Spotted Fever	6	6
Typhus Fever	0	0

ROCKY MOUNTAIN SPOTTED FEVER

The advent of warm weather marks the appearance of the dog tick (Dermacentor variabilis), the principal vector of Rocky Mountain spotted fever in the eastern states. The recognition of this disease in Virginia was established less than ten years ago, but because of the lay publicity which it has received, its presence has cause no little concern in some areas of the State. Therefore, it has been thought wise to review briefly some of the features of this illness as it occurs in Virginia.

An average of approximately fifty cases per year of Rocky Mountain spotted fever is reported to the Virginia Department of Health. The disease has been confined principally to that area of the State which lies east of the Blue Ridge Mountains, with a few cases in the mountain area and only a rare occurrence in the western portion. The cases have occurred chiefly in the late spring and summer (May through September) corresponding to the months of greatest activity of the tick population.

The disease is most prevalent in rural dwellers, with farmers and members of their families comprising the majority of the cases. The group showing the next highest incidence are those whose occupation or pleasure takes them into tick infested woods or fields; foresters, hunters, campers and the like. All ages are attacked and sex is no factor aside from the greater risk of infection of males by virtue of their work.

The onset of the disease may be sudden but usually begins with a prodromal period of a few days in which general lassitude, fatigue, vague aches and pains and dull headache are experienced. This is followed by severe headache in the majority of cases, fever which rapidly rises to as high as 104°-105° in some cases, and occasionally one or more chills. Soreness and pain in the muscles are frequent and a definite stiffness of the neck and back is observed in a fair number of instances. Prostration may be marked and restlessness and insomnia are not infrequent. Delirium, disorientation, or coma may occur in the severe cases. Constipation frequently is observed, and occasionally abdominal tenderness. Bronchitis with scant to non-productive cough is common. The characteristic rash usually appears between the second and sixth day, and begins first on the ankles and wrists and spread centrally to the legs, arms, back, chest and occasionally to the face and scalp. The palms of the hands and soles of the feet may be involved. The rash is at first pink or brownish pink and macular in character, becoming more pronounced in color and, finally, definitely petechial in some cases. When well established, the rash may appear distinctly purple-red. Confluence of lesions has been reported and necrosis of the skin has been observed in severe cases.

The pulse usually is low in proportion to the temperature at the onset, but may become unusually fast, particularly in the terminal stages of the severe cases. The course of the disease usually extends over a two- to three-week period with the temperature subsiding by lysis.

The leukocyte count is of no particular value as an aid to diagnosis. An agglutination reaction with Proteus X2 and X19 is frequently exhibited by the patient's blood serum by the end of ten to fourteen days of the illness.

The case fatality rate observed in Virginia has been approximately twenty to twenty-five per cent. As in other acute infections, the rate is highest in very young children and older adults.

A definite history of tick bite can usually be elicited in about 50 per cent of the cases though the

exact date the bite occurred is not, as a rule, definitely established except in a few of the cases.

The diagnosis of Rocky Mountain spotted fever when a definite tick bite history is given and when the characteristic eruption has appeared is usually simple. The disease must be differentiated from endemic typhus, in which the rash begins centrally and spreads peripherally, in contrast to the peripheral origin of spotted fever. Typhus occurs most often in the colder months, and affects urban dwellers chiefly. Before the appearance of the rash, spotted fever may be mistaken for serebrospinal meningitis or poliomyelitis, because of the frequent occurrence of stiff neck and back and pain and soreness in the muscles. The presence of a slow pulse with high fever may erroneously suggest typhoid fever.

Prevention of Rocky Mountain spotted fever is concerned with (1) avoidance of areas known to be tick infested, (2) removal of ticks from body before they have become attached, (3) vaccination.

The use of clothing that will prevent ticks from gaining access to the body is advisable for those who must necessarily work in tick infested areas. A thorough examination of the body twice a day, at noon and upon retiring, is advisable even when protective clothing has been used. Particular attention should be given the hairy areas. Campers, hunters and other pleasure seekers should avoid old roads and paths with dense undergrowth and overhanging bushes. The best camp site is one where there is little or no low vegetation.

A specific vaccine prepared in the United States Public Health Service laboratories has proven of value in the prevention of Rocky Mountain spotted fever. The vaccine is prepared from the tissues of infected ticks and is given in two doses of 2 cc. each at five-day intervals. The immunity conferred is considered to last approximately one year. The use of this vaccine in Virginia has been confined principally to those persons whose occupation brings them into frequent contact with areas heavily infested with ticks. The vaccine is distributed to physicians upon request without charge. A small supply is kept in the Virginia Department of Health. The vaccine is of no value in the treatment of the disease, and its use for this purpose is ill-advised.

Woman's Auxiliary to the Medical Society of Virginia

President—MRS. JAMES B. STONE, 15 Maxwell Road, Richmond.
President-Elect—MRS. HAWES CAMPBELL, Venter.
Corresponding Secretary—MRS. LAWRENCE O. SNEAD, 5307 New Kent Road, Richmond.
Recording Secretary—MRS. JAMES K. HALL, 3011 Seminary Avenue, Richmond.
Treasurer—MRS. REUBEN F. SIMMS, 2502 Hawthorne Avenue, Richmond.
Chairman of Publicity—MRS. FRED J. WAMPLER, 4103 Forest Hill Avenue, Richmond.

Richmond Auxiliary.

On May 6 the Auxiliary to the Richmond Academy of Medicine, under the direction of the co-chairmen, Mrs. Lee Sutton, Jr. and Mrs. J. K. Hall, sponsored a card party in the Tea Rooms of Miller & Rhoads Department Store which was well attended and which netted the Auxiliary a substantial sum for its philanthropic work.

At a meeting of the Executive Committee on May 17, to take care of interim business, $150.00 was sent the Crippled Children's Hospital to take care of our three beds there for the year, and $36.50 to the State Fund for the T.B. bed at Blue Ridge Sanatorium. Our local contribution to this latter fund each year so far has been a memorial to Mary Gray Hodges. Mrs. Hodges was a past president of the local organization, helped to organize the State Auxiliary and was also a past president of this. She gave largely of time and energy to Auxiliary work and was always enthusiastic about its progress.

The chairmen of the standing committees serving Richmond this year are:

MEMBERSHIP	Mrs. J. K. Hall
PROGRAM	Mrs. J. B. Stone
PRESS AND PUBLICITY	Mrs. Fred J. Wampler
PHILANTHROPIC	Mrs. Lee Sutton, Jr.
SOCIAL	Mrs. Joseph Bear
PHONE	Mrs. Reuben F. Simms
HYGEIA	Mrs. Marshall P. Gordon, Jr.

(MRS. F. J.) REBECCA C. WAMPLER,
Chairman, Press and Publicity.

Periodic Health Examination.

"One out of six applications for life insurance is declined or postponed. The annual health audit will detect albumin or sugar, high blood pressure, slight cardiac disorder, incipient tuberculosis, be-

ginning neoplasm, and any and everything else. Your family physician will do the rest. Why does it profit a person to be an ostrich with his ailments, or like the Spartan youth, to hide a disease until it gnaws out his vitals? Get the disease before it gets you. Get it early. Get it before you think you have it.

"People have too long had such faith and confidence in their physician that they think he can cure anybody who has not been dead for over three days. The profession admires the faith of their clientele, but dislikes to be put in such superlative and unequal tests.

"If elevators are inspected regularly, why not one's mouth and teeth? If a boiler must be examined regularly, why not your heart and lungs? You have tested the brakes on your car, why not your kidney function? You have your watch regulated, but not your diet. You have your batteries charged but let your weight run down from disease.

"Should the most complex and wonderful machinery in the world, that is not made with hands, be allowed to become broken or impaired, to corrode or degenerate? Neglect your household if you must, neglect your bridge and golf if you can, neglect your husband if your dare, but don't neglect your physician and a yearly physical examination and health inventory on your birthday."

—*National Handbook.*

The above should be a bit of timely advice to all, but particularly to us as doctors' wives. We should make full use of the opportunity and privilege of keeping ourselves and our families healthy and thereby set a good example to our neighbors and the general public, as well as enjoy life to the fullest ourselves.

R. C. W.

Truth About Medicine

In addition to the articles previously enumerated the following have been accepted by the Council on Pharmacy and Chemistry of the American Medical Association:

Chas. C. Haskell & Co., Inc.

Sulfanilamide Tablets, 5 grains.

Lederle Laboratories, Inc.

Antipneumococcic Serum Types V and VII (Lederle) Refined and Concentrated.

Antipneumococcic Serum Types IV and VIII (Lederle) Refined and Concentrated.

Eli Lilly & Co.

Ampoules Ephedrine Sulfate—Lilly, 1 cc., 0.025 Gm.

Parke, Davis & Co.

Theelin

Ampules Theelin Aqueous, 1 cc.

Ampules Theelin in Oil, 1 cc.

Vaginal Suppositories Theelin.

Theelol

Kapseals Theelol, 0.06 mg.

Kapseals Theelol, 0.12 mg.

New and Nonofficial Remedies

The following products have been accepted by the Council on Pharmacy and Chemistry of the American Medical Association for inclusion in New and Nonofficial Remedies:

Cevitamic Acid—P. D. & Co.—A brand of cevitamic acid —N. N. R. (New and Nonofficial Remedies, 1938, p. 480). It is supplied in the form of tablets, 25 mg. Parke, Davis & Co., Detroit, Mich.

Cheplin's Epinephrine Hydrochloride Solution (New and Nonofficial Remedies, 1938, p. 232), 1:1,000, 10 cc.—Marketed in rubber stoppered vials for parenteral administration. Cheplin Biological Laboratories, Syracuse, N. Y.

Cheplin's Epinephrine Hydrochloride Solution (New and Nonofficial Remedies, 1938, p. 232); 1:1,000, 30 cc.—Marketed in rubber stoppered vials for parenteral administration and in screw cap vials for topical administration. Cheplin Biological Laboratories, Syracuse, N. Y. (*J. A. M. A.*, May 28, 1938, p. 1837.)

Propaganda for Reform

Viosterol and Psoriasis.—Cedar and Zon (Pub. Health Rep. 52:1580 (November 5), 1937) administered massive doses of viosterol without local treatment of the lesions, dietary adjustment or any other therapeutic measure. A series of fifteen patients from thirty to fifty years of age with chronic widespread psoriasis were given from 300,000 to 400,000 units of vitamin D as viosterol. Eleven of the fifteen subjects showed complete involution of the psoriasis within six to twelve weeks' time. At the end of the period of treatment, three patients showed incipient symptoms of excessive vitamin D dosage. All the subjects exhibited an elevation in the level of blood calcium. There was a recurrence in some of the patients, though the degree of severity was much less than originally observed. Although the proposed treatment would appear to be safe, the authors suggest not only that there may be a smaller effective dose of viosterol but also that certain accompanying products of the irradiation of the ergosterol may be the potent factor. (*J. A. M. A.*, January 8, 1938, p. 133.)

Eleven Deaths From A Cancer Treatment.—In October, 1935, when the "Ensol" treatment was launched from Kingston, Ont., with what appeared to be carefully planned publicity in the newspapers, *The Journal* issued a warning to the effect that the product was being developed under uncontrolled conditions and that its exploitation would inevitably lead to grief for those concerned. Nevertheless a considerable number of doctors in various parts of the United States have used a product of this type in the treatment of cancer and it is obvious that one at least has

come to grief. It seems that the Biochemical Research Foundation of the Franklin Institute of Philadelphia prepared the product called ℞ or Rex, which caused the deaths of eleven patients in Orlando, Fla. It seems likely that batch 152 was prepared on a Friday, some of it permitted to stand over Saturday and Sunday, and then sterilized on Monday. If the tetanus organism was present in the product it would have had two days in which to develop the toxin, so that when the product was sterilized on Monday a sufficient amount of tetanus toxin was present to cause death. These possibilities remain to be confirmed by more evidence—but certainly enough evidence is available to warrant the suggestion. At present there are being exploited to the American people a half dozen or more treatments of cancer that are in no way established as actually of value in the treatment of that condition. Enough is now known about the nature of cancer to indicate that the value of a cancer remedy cannot be established by sending it at random to physicians scattered all over the country, who use it in practice for a fee. The development, exploitation and promotion of "Ensol" and of its progeny "℞" have been unscientific, unethical and unwarranted. (*J. A. M. A.*, April 9, 1938, p. 1194.)

Book Announcements

New Books.

The following are recent acquisitions to the Library of the Medical College of Virginia, which are available to our readers, the only cost being return postage:

Abbott, M. E.—Atlas of Congenital Cardiac Disease.
Association for Research in Nervous and Mental Disease—Pituitary Gland.
Barcroft, J.—The Brain and Its Environment.
Coward, K. H.—Biological Standardization of Vitamins.
De Kruif, P.—The Fight for Life.
Dobzhansky, T.—Genetics and the Origin of Species.
Hadfield, G. & Garrod, S. P.—Recent Advances in Pathology.
Hill, L. & Ellman, P.—Rheumatic Diseases.
History of the American Physiological Society.
Olmsted, J. M. D.—Claude Bernard: Physiologist.
Tauber, H.—Enzyme Chemistry.
U. S. Army—Laboratory Methods of the U. S. Army.
Watson, L.—Hernia.

Manual of the Diseases of the Eye. For Students and General Practitioners. By CHARLES H. MAY, M. D., Consulting Ophthalmologist to Bellevue, Mt. Sinai and French Hospitals, New York, etc. Fifteenth Edition Revised with the Assistance of CHARLES A. PERERA, M. D. Baltimore. William Wood and Company. 1937. Octavo of v-498 pages. With 376 Original Illustrations Including 25 Color Plates, with 78 Colored Figures. Cloth. Price, $4.00.

The fifteenth edition is a thorough revision of this well known manual. In this Dr. May was assisted by Dr. C. A. Perera.

This volume has been brought up to date, with additions and improvements, but without increasing the size.

The chapters on the ocular manifestations of general disease and on the ophthalmoscope have been rewritten. Additions which have been incorporated include those dealing with dinitrophenol cataract, inclusion bodies, inclusion blennorrhoea, acetylcholine therapy, "floaters", gonioscopy, pontocaine, recumbent spectacles, and polaroid glass. Changes have been made in the descriptions of operations upon the lids and in those dealing with the newer operations for detachment of the retina.

P. W.

Information for Expectant Mothers. Metropolitan Life Insurance Company. New York. Copyright 1937. Pamphlet of 48 pages. This pamphlet and another "The Baby" may be obtained from the Metropolitan Life Insurance Company. Booklet Department 5-R-38, One Madison Avenue, New York City.

Hospital Service in the United States. 1938. Seventeenth Presentation of Hospital Statistics by the Council on Medical Education and Hospitals of the American Medical Association. The 1937 Census of Hospitals. Hospitals Registered by the American Medical Association. Reprinted from the Hospital Number of the *Journal of the American Medical Association*, March 25, 1938. Pamphlet of 93 pages. Price, 50 cents.

The Role of the Social Worker in a Prevention of Blindness Program. By LEWIS H. CARRIS, Managing Director, and ELEANOR BROWN MERRILL, Associate Director, National Society for the Prevention of Blindness. Publication 258. National Society for the Prevention of Blindness, New York, N. Y. Pamphlet of 8 pages. Price, 5 cents.

Glaucoma. By M. CARL WILENSKY, M. D., Assistant Professor of Ophthalmology, Tulane University, New Orleans, La. Publication 256. National Society for the Prevention of Blindness. New York, N. Y. Pamphlet of 16 pages. Price, 15 cents.

XUM

Virginia Medical Monthly

Founded by LANDON B. EDWARDS, M. D., April, 1874
Owned by MEDICAL SOCIETY OF VIRGINIA since November, 1919
WYNDHAM B. BLANTON, M. D., *Editor*
AGNES V. EDWARDS, Richmond, *Business Manager*

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All correspondence regarding editorial matters, articles, advertisements, subscription rates, etc., should be addressed to the Monthly, 1200 East Clay Street, Richmond, Va.
This journal is not responsible for the opinions and statements of its contributors.
All advertisements are received subject to the approval of the Council on Pharmacy and Chemistry of the American Medical Association.
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VOLUME 65 JULY, 1938 No. 7

Editorial

Why Our Papers Are Rejected.

There are probably few medical authors who have not had a paper rejected by the *Journal of the American Medical Association.* If any of them have, they may find the explanation in a useful and condensed hand book just off the press of the Association entitled "Medical Writing, the Technique and Art", by none other than the editor Morris Fishbein himself. It may be some comfort to know that three-fourths of all papers received by the *Journal of the A. M A.* are rejected. The chief reason for this large number is lack of space. The number of original volunteered manuscripts which can be published yearly in the *Journal* varies between five hundred and six hundred. The number of manuscripts annually received by the *Journal* varies between two thousand and three thousand. When you send a paper to the *Journal,* all else being equal, you have a one in four chance of its being accepted. If the subject of the paper is one on which there is a plethora of material and has been recently overworked, such as for example foci of infection, the author is very likely to have the manuscript returned in short order bearing one of the polite rejection sheets. If you are fond of mental gymnastics and produce a purely theoretical paper, the paper is usually returned with the suggestion that an ounce of fact is worth a ton of theory. If you have delivered an address before a medical society, it is apt to be prolix, wordy, and hastily assembled—perhaps just a pot-boiler gotten together for reading rather than for printing. The fate of such a paper is likely to be the same. The editor thinks that "a manuscript that is fit to read is sometimes fit to print, but that a manuscript fit to print is always fit to read." Dr. Fishbein does not like long papers and he devotes a whole chapter of his book to showing how by careful revision most papers can be reduced by at least twenty-five per cent.

Medical Solecisms.

In one of his chapters Fishbein expatiates at length upon the barbarisms of medical writing, citing from actual papers that have been submitted for publication. The Editor of the *Journal of the American Medical Association* does not like for his contributors to describe patients who have "no temperature", or for doctors to "inject patients with a drug". He prefers to have them inject cadavers and to remember that the verb is transitive and requires an object. He does not wish us to say "a malignancy was removed" or to use abstract words in a concrete sense as "the cytology was normal", or "there was no pathology", or "surgery seemed the best treatment". He wishes us to remember that sphenoid and ethmoid are nouns and that their use as adjectives in the phrase "sphenoid and ethmoid sinuses" is not allowable. He does not like the "right heart" or "the left chest" or a "cardiac diet", because the adjective in each case modifies the wrong word. He

shudders a bit when the "patient has *an* anemia" and his discomfort is worse when per cent is used as a noun or when a surgeon says "I operated this patient". He won't allow a pathologist or those who quote him to report "autopsy findings" or "what was found at operation". He will allow "autopsy observations" or what was seen, noted, or observed at operation. The editor is supported by every dictionary in the English language when he objects to physicians reporting that a "patient developed a tumor". Nor will he allow a contributor to say he "took a biopsy". Instead he must say "he excised a specimen for biopsy". His editorial red pencil goes straight through "we", "I", and "the author" when used indiscriminately in the same article and he gets the creeps when a writer uses such over worked phrases as the "microscope showed", "throws light", "points out", and "symptom complex". He is averse to the superlatives "quite", "marked", and "great", and a copy in which the position of a previous or succeeding statement is referred to as "above" or "below" is certain to be blue penciled.

The Essence of a Good Medical Paper.

Fishbein's handbook should find a place on the desk of every medical author. In his chapter on style he enjoins simplicity and clearness. In his discussion of subject and material he gives admirable advice, particularly elaborating the preparation of the now much neglected case report. He discusses the detailed construction of the manuscript, title, words and phrases, spelling, capitalization, abbreviations, numbers, proper usage in the matter of pharmaceutical products and prescriptions, and how to secure a bibliography. With considerable detail he goes into the mechanical preparation of manuscripts, illustrations, charts, and tables. He concludes with emphasis upon the importance of revision, telling us that Sir William Osler always made at least three drafts of a manuscript, Sir Clifford Albutt four, and Anatole France eight, but leading us to believe that he rather agrees with Trelease and Yule who recommend ten before submitting the result to the printer.

But for Our Tubules.

"For the formation of 1 cc of urine per minute, more than 100 cc. of fluid are separated from the blood—in this separated fluid are contained, in twenty-four hours, nearly one-half pound of glucose, or bicarbonate, and two pounds of salt."

In these words Alfred N. Richards recently summarized the astonishing results of glomerular filtration. By a simple calculation it is seen that the daily combined output of all the glomeruli amounts to approximately 100,000 cc. Expressed differently, if the human kidneys contained only glomeruli and nothing but this filtration process went on, the average individual would pass twenty-five gallons of urine a day. From this it is readily seen what an important function the tubules perform in reabsorption.

The fascinating story of the acquisition of knowledge concerning the function of the kidneys, which began with Bellini and Malpighi in the seventeenth century and reached an impasse in the general debate over the relative virtues of Bowman's and Ludwig's theories two hundred years later, has been steadily unfolded since 1921 through the epoch-making work of Richards and his collaborators. There remains yet much to be done not only in a study of the selective absorption and secretion activities of the cells of the various parts of the uriniferous tubules but in the effort to adapt these experimental findings in the frog and Necturi to the human kidneys.

Department of Clinical and Medical Education of the Medical Society of Virginia

Obstetrics and Gynecology.

During the month of June Dr. Shamburger has conducted a postgraduate course in the Valley of Virginia, holding meetings at Staunton, Harrisonburg, Woodstock, and Front Royal. A meeting scheduled at Luray was discontinued due to poor attendance. Those attending at other points were:

STAUNTON

Dr. Chas. Bickley Fox — Dr. Chas. Wm. Rodgers
Dr. M. J. Payne — Dr. Geo. Stone
Dr. W. C. Roller

HARRISONBURG

Dr. Howard Armstrong — Dr. M. S. Foster
Dr. Ashby C. Byers — Dr. B. W. Nash
Dr. F. L. Byers — Dr. H. A. Stratton
Dr. J. J. Waff

WOODSTOCK

Dr. F. C. Downey — Dr. E. L. Hopewell
Dr. David O. Foley — Dr. Harold Miller
Dr. R. C. Fravel — Dr. J. M. Winkfield

FRONT ROYAL

Dr. C. H. Armentrout — Dr. L. F. Hansbrough
Dr. O. W. Carper — Dr. J. McI. Jackson
Dr. E. L. Grubbs — Dr. D. M. Kipps

Dr. Shamburger with the completion of this circuit finishes the service for which he was engaged by the Department of Clinical and Medical Education. Since his employment in July, 1936, he has conducted courses in Obstetrics and Gynecology in all sections of the State. His work has been highly satisfactory and pleasing to the doctors wherever he has been. We have been fortunate to have his services and extend our best wishes to him in his new work. In a future issue we plan to submit a summary of his valuable services to the doctors of the State.

Pediatrics.

On May 27, Dr. Hightower completed his circuit in the Mid-Tidewater section. The following doctors attended his course at the points indicated:

NEWPORT NEWS

Dr. Wm. P. Dickerson — Dr. E. W. Buckingham
Dr. Ernest C. Downing — Dr. Paul Hogg
Dr. Floyd W. Green — Dr. C. P. Jones
Dr. R. A. B. Lloyd — Dr. Thomas C. Lawford
Dr. I. B. McEachin — Dr. J. W. Sayre
Dr. Russell E. Reed — Dr. T. D. Walker
Dr. A. T. Scott
Dr. G. B. D. Stephens
Dr. W. D. Young

WILLIAMSBURG

Dr. B. I. Bell — Dr. F. R. Person
Dr. C. E. Holdbery — Dr. Slemons (Visitor from Harvard)
Dr. E. B. Kilby — Dr. Albert M. Sneed
Dr. T. E. Painter — Dr. J. R. Tucker
Dr. James R. Parker

NORFOLK

Dr. A. H. Buck — Dr. G. H. Francis
Dr. S. T. Canaday — Dr. J. D. Jackson
Dr. C. R. S. Collins — Dr. S. I. Moone

HAMPTON

Dr. Burl Bassette — Dr. H. D. Howe
Dr. Herbert T. Berwald — Dr. B. E. Hunt
Dr. A. C. Davis — Dr. P. J. Parker
Dr. Charles Faber — Dr. W. P. Smith
Dr. W. H. Howard — Dr. O. W. Ward
Dr. R. H. Wright, Jr.

In June Dr. Hightower has been engaged in conducting a course in the Clinch Valley area. Meetings are being held at Coeburn, Norton, Stonega, Appalachia, Pennington Gap, and Gate City. The interest and attendance in this area has been marked and much better than in the past. Arrangements are being made for a circuit in the northern part of the Clinch Valley following the present course.

Internal Medicine.

Plans are being made for a course in Internal Medicine to be conducted in the area of the Southwestern Virginia Medical Society during the latter part of July. Meetings will probably be held at Abingdon and Wytheville. The instructors for this course will come from the faculty of the University of Virginia Medical School. A similar course may be held in other sections upon the request of local societies.

GEO. B. ZEHMER,
Executive Secretary.

Proceedings of Societies

A Correction.

We regret that in the June issue of the MONTHLY, the new officers of the Roanoke Academy of Medicine were incorrectly stated. They should be: President, Dr. W. W. S. Butler; vice-presidents, Drs. L. G. Richards and C. A. Young; and secretary-treasurer, A. C. Davis.

Norfolk County Medical Society.

At the annual meeting of this Society in June, Dr. William B. Newcomb succeeded to the presidency, and Dr. Harvie S. Baker was named president-elect. Dr. Benjamin A. Doggett was elected vice-president and Dr. Lockburn B. Scott was re-elected secretary-treasurer for his twentieth successive year. All of-

ficers are of Norfolk. Delegates and alternates were also selected for the Danville meeting of the State Society.

At this meeting, Dr. Scott signified his intention of spending the summer on a visit to his daughters in Winnipeg, Canada, and asked that Dr. A. Brownley Foster be appointed acting secretary-treasurer for the period of his absence, which request was granted. Dr. Scott will return in September.

Charlotte County Medical Society.

At a recent meeting of this Society, the following officers were elected: President, Dr. C. M. Nicholson, Charlotte C. H.; vice-president, Dr. J. B. Bailey, Keysville; and secretary, Dr. R. B. Cralle, Drakes Branch.

Hanover County Medical Society.

At a meeting of this Society, the latter part of May, the following officers were elected: President, Dr. Hawes Campbell, Jr., Hanover; vice-president, Dr. Edwin Vaughan, Ashland; and secretary-treasurer, Dr. Frank L. Hughes, Ashland. Drs. Edwin Vaughan, I. K. Redd, and T. E. Stanley were named as a committee to work up details for hospitalization of Hanover indigents.

The Lynchburg Academy of Medicine

Held its regular monthly meeting, June 6, with Dr. Sam Oglesby, vice-president, presiding. Delegates and alternates to the State Society meeting in Danville were elected. Dr. J. Shelton Horsley, Richmond, presented a paper on "Carcinoma of the Stomach", accompanied with case records and lantern slides.

The next meeting of the Academy will be held in September.

The Fourth District Medical Society

Held its annual meeting in Blackstone, May 24. Dr. C. V. Montgomery, South Hill, was elected president, succeeding Dr. J. L. Hamner, Mannboro. Other officers are: vice-presidents, Dr. C. G. O'Brien, Appomattox and Dr. N. P. Snead, Cartersville; secretary-treasurer, Dr. C. E. Martin (re-elected), Emporia; and Dr. Wright Clarkson, Petersburg, chairman of the steering committee. The standing committees were re-appointed. Dr. William Feild, Petersburg, was elected to membership.

Dr. W. B. McIlwaine, Petersburg, was nominated to the State Society, to succeed the late Dr. Fletcher Wright, Sr., as representative of the fourth district on the State Board of Medical Examiners, this nomination to be presented to the House of Delegates at its next meeting.

The Medical Association of the Valley of Virginia

Held its regular meeting on May 26 in Harrisonburg, under the presidency of Dr. R. P. Bell, of Staunton. The following program was presented: The Present Status of Coronary Disease by Dr. B. S. Yancey, Harrisonburg; The Relation Between Pre-Natal Care and Obstetrical Mortality by Dr. L. M. Allen, Winchester; Fractures of the Neck of the Femur by Dr. R. V. Funsten, University; Mesenteric Adenitis by Dr. J. M. Emmett, Clifton Forge; and Sulfanilamide with report of the Treatment of a Case of Puerperal Septicemia by Dr. Alex F. Robertson, Jr., Staunton.

The Southside Virginia Medical Association

Held its regular quarterly meeting at the Central State Hospital, Petersburg, on June 14. Dr. W. J. Ozlin, South Hill, is president, and Dr. R. L. Raiford, Franklin, secretary. The following program was presented: Dr. Herbert C. Jones, Petersburg, "A Presentation of Urological Cases"; Dr. Roy K. Flannagan, Richmond, "Pioneering in Health 1908-1910"; Dr. J. S. Horsley, Jr., Richmond, "Peritonitis Treated with Sulfanilamide"; Dr. James Asa Shield, Richmond, "Insulin Therapy in Mental Diseases"; and Dr. Meade Edmunds, Petersburg, "Ocular Manifestations of Vitamin Deficiency." Dr. J Bolling Jones of Petersburg reported an interesting case of Traumatic Pneumothorax from an automobile accident.

A business session was held following the scientific program, and the meeting was closed with a dinner tendered by Dr. Henry and his staff at the hospital.

The Staunton Academy of Medicine,

At its meeting on May 24, elected the following new officers: President, Dr. Joseph Alexander; vice-president, Dr. Guy R. Fisher; and secretary-treasurer, Dr. Charles W. Putney.

The Academy recommended to the City of Staunton that they renew their efforts to obtain an appropriation from the proper authorities for enclosing the sewage system and establishing a satisfactory sewage disposal plant.

XUM

The Virginia, Maryland and District of Columbia Medical Society

Held its semi-annual meeting at the Manor Club, Norbeck, Md., on May 25, under the presidency of Dr. James Gannon, Washington. Among those presenting papers were Drs. W. Calhoun Stirling, W. Dabney Jarman, Tomas Cajigas, Robert Sterling McGrath, Edward M. Pickford, and Daniel D. V. Stuart, Jr, all of Washington, and Dr. William O. Bailey, Leesburg.

At the business meeting, Dr. M. B. Hiden, Warrenton, was unanimously elected president for the coming year

News Notes

Commencement Exercises of Virginia Medical Schools.

MEDICAL COLLEGE OF VIRGINIA

The Centennial Session of the Commencement exercises of the Medical College of Virginia were held June 4 to 7. The functions were opened with the student night dance at the John Marshall Hotel. On Sunday, the commencement sermon was given by the Right Reverend Beverley D. Tucker, D. D., Rector of St. Paul's Church.

Alumni registration began on Monday, and more than two hundred attended the business meeting and luncheon. The annual banquet was held that night, with Dr. R. W. Miller, president, presiding. The following officers were elected for the ensuing year: president, Dr. F. P. Fletcher, Richmond; vice-presidents, Dr. Meade Edmunds, Petersburg; Dr. A. M. Wash, Richmond; Mr. D. D. Gray, Sr., Norfolk; and treasurer, Dr. T. Dewey Daivs, Richmond.

The Centennial Program was held at St. Paul's Church on Tuesday, the 7th. One hundred and thirty delegates, representing as many universities and organizations throughout the country, were in the academic procession, which comprised three hundred and fifty persons. The principal address was given by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard University Medical School, Boston. Following this meeting, a buffet luncheon was served the Board of Visitors, Centennial Delegates, Alumni, Faculty, and Senior Classes.

The final exercises were held at the Mosque, with the Commencement address being made by James Rion McKissick, President of the University of South Carolina. The Honorary degree of Doctor of Science was conferred upon Dr. James Carroll Flippin, Dean of the Medical School of the University of Virginia.

There were one hundred and thirty-nine graduates from the four schools of the college—seventy-six in medicine, sixteen in dentistry, twenty-one in pharmacy, and twenty-six in nursing. Thirty graduates received commissions in the Officers' Reserve Corps —twenty-one in the Medical Corps and nine in the Dental Corps.

Following are the graduates in medicine, with hospital appointments:

HOSPITAL DIVISION, MEDICAL COLLEGE OF VIRGINIA, RICHMOND—Drs. Ernest Linwood Bagby, Richmond; Hubert Dinwiddie Crow, Richmond; James Thomas Gill, Richmond; Gordon Douglas Hall, Dumbarton; Thomas Holt, Warrenton, N. C.; John Tallman Jarrett, Dunbar, W. Va.; William Herbert McCall, Richmond; Paul James Nutter, Clarksburg, W. Va.; and Marvin Everett McRae, Richmond.

STUART CIRCLE HOSPITAL, RICHMOND—Drs. Robert Franklin Bell, Butte, Mont.; Henkel Moser Price, Richmond; William Parker Terry, Burkeville; and George Harrison Williams, Petersburg.

ST. LUKE'S HOSPITAL, RICHMOND—Drs. Edwin Clinton Bryce, II, Richmond; Garland Dyches, Buffalo Ridge; and Hazael Joseph Williams, Richmond.

JOHNSTON-WILLIS HOSPITAL, RICHMOND—Drs. William Henry Copley, Richmond; George Simeon Fultz, Jr., Butterworth; and Carl Scott Lingamfelter, Dumbarton.

UNIVERSITY OF VIRGINIA HOSPITAL, CHARLOTTESVILLE—Dr. Bathurst Browne Bagby, Jr., Richmond.

ST. VINCENT'S HOSPITAL, NORFOLK—Dr. Ralph Boyd Blowe, Boykins.

NORFOLK GENERAL HOSPITAL, NORFOLK—Dr. William Hart Woodson, Newport News.

LEWIS-GALE HOSPITAL, ROANOKE—Drs. Earle Bailey Morgan, Dublin; and Darwin Elbert Smith. Richmond.

ELIZABETH BUXTON HOSPITAL, NEWPORT NEWS —Dr. Claudio Rodriguez Arce, Alajuela, Costa Rico.

CHARLESTON GENERAL HOSPITAL, CHARLESTON, W. VA.—Drs. Julius Lyons Berkley, Charleston, W. Va.; Howard Dewey Booth, Clendenin, W. Va.; Samuel Stuart DuPuy, Beckley, W. Va.; and James Eugene Grace, Morgantown, W. Va.

CHESAPEAKE AND OHIO HOSPITAL, HUNTINGTON, W. Va.—Drs. Robert Morris Ferrell, Lewisburg, W. Va.; Liskie Jay Moore, Parkersburg, W. Va.; and John Franklin Morris, Huntington, W. Va.

OHIO VALLEY GENERAL HOSPITAL, WHEELING, W. VA.—Drs. Jacob Camden Huffman, Webster Springs, W. Va.; and Herbert Grayson Ruffin, Richmond.

HINTON HOSPITAL, HINTON, W. VA.—Dr. Albert William Holmes, Lowell, W. Va.

HIGHSMITH HOSPITAL, FAYETTEVILLE, N. C.—Dr. David Melvin Cogdell, Fayetteville, N. C.

CITY MEMORIAL HOSPITAL, WINSTON-SALEM, N. C.—Dr. Will Hardee Lassiter, Jr., Smithfield, N. C.

JAMES WALKER MEMORIAL HOSPITAL, WILMINGTON, N. C.—Dr. Walter Glenn Lewis, Elon College, N. C.

REX HOSPITAL, RALEIGH, N. C.—Drs. Marvin Bailey Poole, Raleigh, N. C.; Jack Gregory Tillery, Halifax, N. C.; and Annie Louise Wilkerson, Raleigh, N. C.

CONEMAUGH VALLEY MEMORIAL HOSPITAL, JOHNSTOWN, PA.—Dr. Leon J. Anson, Bronx, N. Y.

PHILADELPHIA GENERAL HOSPITAL, PHILADELPHIA, PA.—Dr. Earl Shelbourne Scott, Welch, W. Va.

EPISCOPAL HOSPITAL, PHILADELPHIA, PA.—Dr. Edward George Sharp, Philadelphia, Pa.

QUEEN'S GENERAL HOSPITAL, JAMAICA, L. I., N. Y.—Dr. Russell Neff Carrier, Richmond.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL, NEW YORK, N. Y.—Dr. Charles Fleetwood James, Jr., Appomattox.

CONEY ISLAND HOSPITAL, BROOKLYN, N. Y.—Dr. Sydney Loeb Lang, Brooklyn, N. Y.

JOHNS HOPKINS HOSPITAL, BALTIMORE, MD.—Dr. Haye Woodrow Caldwell, Athens, W. Va.

U. S. MARINE HOSPITAL, NORFOLK—Drs. Phillips Lester Claud, Portsmouth; and Norman Elwood King, Haynesville.

U. S. MARINE HOSPITAL, BALTIMORE, MD.,—Dr. Albert John Russo, Wilkes-Barre, Pa.

GALLINGER MUNICIPAL HOSPITAL, WASHINGTON, D. C.—Dr. Albert Anthony Kossove, New York, N. Y.

CITY HOSPITAL, CLEVELAND, OHIO—Dr. Evelyn Mary Ball, Huntington, W. Va.

BOSTON CITY HOSPITAL, BOSTON, MASS.—Drs. Archie Arthur Hoffman, Chelsea, Mass.; and William Taliaferro Thompson, Jr., Richmond.

PETER BENT BRIGHAM HOSPITAL, BOSTON, MASS. —Dr. Sidney Grey Page, Jr., Richmond.

DUVAL COUNTY HOSPITAL, JACKSONVILLE, FLA. —Dr. Raymond Scrivener Blackman, Vienna.

TAMPA MUNICIPAL HOSPITAL, TAMPA, FLA.—Dr. Ira Chenault Evans, Winchester, Ky.

NASHVILLE GENERAL HOSPITAL, NASHVILLE, TENN.—Dr. John Washington Clark, Washington.

BARONESS ERLANGER HOSPITAL, CHATANOOGA, TENN.—Drs. Howard Garnett Snead, Tappahannock; and Paul Clarence Soulsby, Pratt, W. Va.

HILLMAN HOSPITAL, BIRMINGHAM, ALA.—Dr. Julius Frederick Chairsell, Jr., Richmond.

RECEIVING HOSPITAL, DETROIT, MICH.—Dr. Alexander Miller Earle, Jr., Stovall, N. C.

HENRY FORD HOSPITAL, DETROIT, MICH.—Dr. Eugene Orwin Wright, Clarksburg, W. Va.

JERSEY CITY MEDICAL CENTER, JERSEY CITY, N. J.—Dr. Stanley John Fink, Philadelphia, Pa.

NEWARK CITY HOSPITAL, NEWARK, N. J.—Dr. Otto Selick Steinreich, Newark, N. J.

BARNERT MEMORIAL HOSPITAL, PATERSON, N. J. —Dr. Margaret Brinkerhoff Williams, Paterson, N. J.

NEW BRITAIN HOSPITAL, NEW BRITAIN, CONN. —Dr. Myer Goldschmidt, Hartford, Conn.

STATE OF WISCONSIN GENERAL HOSPITAL, MADISON, WIS.— Drs. Edward Ellis Haddock, Richmond; and Gilman Rackley Tyler, Richmond.

ST. JOSEPH HOSPITAL, LEXINGTON, KY.—Dr. Woodrow Wilson Scott, Williamson, W. Va.

PRESBYTERIAN HOSPITAL, SAN JUAN, PUERTO RICO—Dr. Jose William Dwight Santiago, Mayaguez, P. R.

Others receiving their medical degrees are:

Dr. Ina Clair Hall, Charleston, W. Va.

Dr. Jack Nichols, Los Angeles, Calif.

Dr. George Robert Rosenbaum, Richmond.

Dr. Irving Murray Schor, Brooklyn, N. Y.

XUM

UNIVERSITY OF VIRGINIA

The Department of Medicine of the University held its final exercises in conjunction with those of other departments of the University on June 13, 14 and 15. An outstanding feature of this year's exercises was the one hundredth anniversary of the Alumni Association, this having been organized in 1838 at the close of the fourteenth session of the University. On Alumni day, the Thomas Jefferson Society of Patriarchs was organized with all alumni who were students of the University fifty years ago becoming charter members. Dr. Paul B. Barringer of Charlottesville was named grand patriarch of the new group. Dr. Carrington Williams of Richmond was elected vice-president of the Alumni Society.

The usual social events in connection with Commencement were enjoyed and the new library was dedicated as a memorial to the late Dr. Edwin A. Alderman, former president of the University.

Names of the fifty-six graduates of the Department of Medicine with their hospital appointments follow:

UNIVERSITY OF VIRGINIA HOSPITAL, UNIVERSITY—Drs. DuPont Guerry, III, Greenville, S. C.; Charles Johnston Harkrader, Bristol; Leslie Mac Lisle, Jr., Columbus, Ohio; Philip Pendleton Steptoe, Jr., Shepherdstown, W. Va.; and Robert Hamilton Williams, Olympia, Wash.

UNIVERSITY OF VIRGINIA, DEPARTMENT OF MEDICINE, UNIVERSITY—Dr. Orville Russell Kelley, University.

ST. ELIZABETH'S HOSPITAL, RICHMOND—Drs. Elton Meredith Alrich, Fredericksburg; and Frederick Gaston Woodson, Covington.

MEMORIAL HOSPITAL, RICHMOND—Dr. James Garnett Willis, Remington.

RETREAT FOR THE SICK, RICHMOND—Dr. Edwin Burwell Jones Whitmore, Jr., Petersburg.

ROANOKE CITY HOSPITAL, ROANOKE—Dr. Octavius Lake Huffman, Jr., University.

NORFOLK GENERAL HOSPITAL, NORFOLK—Dr. Joseph Lee Mann, Hampton.

VIRGINIA MASON CLINIC, SEATTLE, WASH.—Drs. John Daniel Call, Richmond; and Jacob Grant Hebble, Newport News.

CLEVELAND CITY HOSPITAL, CLEVELAND, OHIO—Drs. Elizabeth Virdin Barnes, Ivy Depot; and Benjamin Calloway Jones, Jr., Huntington, W. Va.

CINCINNATI GENERAL HOSPITAL, CINCINNATI, OHIO—Drs. William Thomas Moore, Blackridge; and Charles Thomas Nicholson, Jr., Alexandria.

FIFTH AVENUE HOSPITAL, NEW YORK, N. Y.—Dr. Edward Gordon Bell, Jr., Bronxville, N. Y.

RICKERS ISLAND HOSPITAL, NEW YORK, N. Y.—Dr. Charles Nichols Romaine, Jr., Petersburg.

BELLEVUE HOSPITAL, NEW YORK, N. Y.—Dr. John Kirk Train, Jr., Savannah, Ga.

CONEY ISLAND HOSPITAL, BROOKLYN, N. Y.—Dr. Malcolm Foote Sher, New York, N. Y.

LONG ISLAND COLLEGE HOSPITAL, BROOKLYN, N. Y.—Dr. George Henry Stollwerck, University.

STRONG MEMORIAL HOSPITAL, ROCHESTER, N. Y.—Dr. James Bell Black, Jr., Red Springs, N. C.

UNION MEMORIAL HOSPITAL, BALTIMORE, MD.—Drs. Richard Phillips Bell, Jr., Staunton; Thomas Jackson Humphries, Culpeper; and Maynard Putney Smith, Farmville.

U. S. MARINE HOSPITAL, BALTIMORE, MD.—Drs. Murray Cox Brown, Richmond; and Edward Worthington Venning, University.

JOHNS HOPKINS HOSPITAL, BALTIMORE, MD.—Drs. Dabney von Knobloch Moon, Charlottesville; and Charles Lemuel Prince, III, Cheraw, S. C.

SINAI HOSPITAL, BALTIMORE, MD.—Dr. Albert George Schurman, Roanoke.

ST. LUKE'S HOSPITAL, BETHLEHEM, PA.—Dr. Belton Allen Bennett, Jr., Greer, S. C.

ST. FRANCIS HOSPITAL, PITTSBURGH, PA.—Drs. Joseph Coudon, VI, Wheeling, W. Va.; Ivor David Harris, New Philadelphia, Ohio; and William Alfred Mitchell, Newport News.

LANKENAU HOSPITAL, PHILADELPHIA, PA.—Dr. Reverdy Hamlin Jones, Jr., Portsmouth.

WOMAN'S HOSPITAL OF PHILADELPHIA, PHILADELPHIA, PA.—Dr. Charlotte Ellen Swaney, East Radford.

UNIVERSITY OF PENNSYLVANIA HOSPITAL, PHILADELPHIA, PA.—Dr. George Nelms Wise, Jr., Hampton.

ALLENTOWN GENERAL HOSPITAL, ALLENTOWN, PA.—Dr. Lewis Fulton Sprague, Allentown, Pa.

MUHLENBERG HOSPITAL, PLAINFIELD, N. J.—Dr. Charles Nuckols Davidson, Nuckols.

ORANGE MEMORIAL HOSPITAL, ORANGE, N. J.—Dr. Robert Bradford Orr, Asheville, N. C.

WILMINGTON GENERAL HOSPITAL, WILMINGTON, DEL.—Dr. Herbert William Fink, Norfolk.

DELAWARE HOSPITAL, WILMINGTON, DEL.—Dr. Joseph Parker Griffin, Portsmouth.

WATTS MEMORIAL HOSPITAL, DURHAM, N. C.—Dr. John Hill Fitzgerald, Jr., Crewe.

NORWOOD HOSPITAL, BIRMINGHAM, ALA.—Dr. John Ferguson Gayle, Newport News.

EMERGENCY HOSPITAL, WASHINGTON, D. C.—Dr. Joseph Harvey Harris, Brooklyn, N. Y.

DUVAL COUNTY HOSPITAL, JACKSONVILLE, FLA.—Dr. John Harrell Hill, Charlottesville.

TOURO INFIRMARY, NEW ORLEANS, LA.—Dr. John Randolph Kight, Norfolk.

WOMAN'S AND CHILDREN'S HOSPITAL, CHICAGO, ILL.—Dr. Jessie Dinsmore Marsh, Lynchburg.

UNIVERSITY HOSPITAL, IOWA CITY, IA.—Dr. Charles Augustus Mella, Jr., West New York, N. J.

HARPER HOSPITAL, DETROIT, MICH.—Dr. William Shepherd Smith, Catonsville, Md.

ROPER HOSPITAL, CHARLESTON, S. C.—Dr. Horace Gilbert Smithy, Berryville.

ROYAL VICTORIA HOSPITAL, MONTREAL, CANADA—Dr. Francis Record Whitehouse, Lynchburg.

U. S. NAVAL HOSPITAL—Drs. John Burton MacGregor, Afton; and George Leroy Tabor, Jr., Cherrydale.

Advisory Board to Woman's Auxiliary.

Dr. G. F. Simpson, President, has appointed Dr. P. St. L. Moncure of Norfolk as chairman of the Advisory Board to the Woman's Auxiliary of the Medical Society of Virginia, succeeding Dr. Fletcher J. Wright who died recently. He has also named Dr. Frederick Gochnauer of Upperville as a new member of this Committee. Dr. James B. Stone of Richmond is the third member.

Have you Named your Delegates and Alternates to the State Meeting?

Component societies of the Medical Society of Virginia have been asked to report to headquarters office names of their delegates and alternates for the Danville meeting of the State Society. It will be appreciated if those who have not already done so will give this matter their attention promptly.

The Southwestern Virginia Medical Society, in sending their names stated that they allow a flat sum of $10.00 to delegates who attend the State meeting and furnish their secretary a report of the transactions of the House of Delegates. Might not this suggestion increase representation?

Married.

Dr. Louis Philip Bailey of Nathalie and Miss Telia Barner Barksdale of Sutherlin, June 18.

Dr. James Thomas Tucker of Richmond and Miss Katharine Vinyard Huff of Roanoke, June 15.

Dr. William Stone Burton, class of '37, Medical College of Virginia, now of Powhatan, and Miss Dorothy Boyd Archer of Petersburg, in June.

Dr. Homer Earle Ferguson of Richmond and Miss Audrey Bryan Tulloh of Mineral, June 3. Dr. Ferguson graduated from the Medical College of Virginia in 1936 and, after an internship at Grace Hospital this city, located in Richmond for practice.

Dr. James Thomas Gill, of the class of '38, Medical College of Virginia and a son of Dr. and Mrs. Thos. F. Gill of Richmond, to Miss Mary Dinwiddie Crow also of this city, June 25.

Dr. William Garland Talmage of Succasunna, N. J., and Miss Anne Radford Trott of Staunton, May 27. Dr. Talmage is an alumnus of the Medical College of Virginia, class of '31.

Dr. DuPont Guerry, III, class of '38, University of Virginia, Department of Medicine, and Miss Sallie Kennon Williams of Lynchburg, June 14. Dr. Guerry has just received an appointment for internship at the University Hospital.

Personnel News from State Health Department.

Dr. Lonsdale J. Roper, formerly an associate director of the Division of Venereal Disease Control, has been appointed director of the Bureau of Rural Health, and assumed his duties on June 7.

Dr. Edgar C. Harper, director of the Crippled Children's Bureau, in addition to his duties in that capacity, has assumed the supervision of the Department's Tuberculosis Out-Patient Service. This activity formerly was administered jointly by the Bureau of Rural Health and the Bureau of Public Health Nursing.

Dr. G. R. Carpenter, who had been temporarily in charge of the Washington, Bristol City Health Department during the absence of Dr. M. I. Shanholtz, has been appointed health officer of the Dickenson-Scott-Wise Health District, succeeding Dr. J. R. Massie who resigned. The office for this district is located at Norton.

Dr. Charles L. Savage, who has returned from post-graduate study at Johns Hopkins School of Hygiene and Public Health, has been appointed director of the Hanover County Health Department, with headquarters at Ashand.

XUM

In the Peninsula Health District, Dr. J. B. Porterfield has resumed direction at Williamsburg. This district had been under the supervision of Dr. C. L. Riley during Dr. Porterfield's absence while he was pursuing advanced study at Johns Hopkins.

Dr. C. L. Riley has become head of Prince William County Health Department with headquarters at Manassas. Dr. William Y. Garrett has been designated assistant health office for that district.

Dr. Linwood Farley has been re-appointed assistant health officer of the Valley Health District with headquarters at Luray. Dr. Farley for several months had been acting health officer of Hanover County Health Department.

In the Washington-Bristol City Health Department, Dr. M. I. Shanholtz, having recently completed his post-graduate work at Johns Hopkins, has been re-appointed director, with headquarters at Bristol.

American Medical Association.

As we go to press, we learn that Dr. Irvin Abell, prominent physician of Louisville, Ky., succeeded to the presidency, and Dr. Rock Sleyster, Wauwatosa, Wis., was named president-elect of the American Medical Association. Dr. Sleyster has always taken an active part in the work of the A. M. A. and was recently chairman of the Board of Trustees.

St. Louis was named as the 1939 convention city.

Medical Society of the District of Columbia.

At the annual meeting of this Society in May, Dr. William J. Mallory was inducted into office as president, and the following were elected for the coming year: President-elect, Dr. John H. Lyons; vice-presidents, Dr. William T. Gill, Jr., and Dr. David Davis; and Dr. Coursen B. Conklin was re-elected secretary. All officers are of Washington.

Coroner of Lynchburg.

Dr. James A. Wilkins has been appointed coroner for the City of Lynchburg, succeeding Dr. J. Burton Nowlin who served in that office for twenty-one years.

Doctors to Serve Hopewell.

In the June elections, both Dr. D. Lane Elder and Dr. S. B. Perry of Hopewell were elected members of the City Council of that place for another term.

Dr. J. F. Phillips,

Recently of Deltaville, is now on the medical staff of the Central State Hospital, Petersburg.

American Psychiatric Association.

At the meeting of this Association, held in San Francisco, June 6-10, Dr. Richard H. Hutchings, Utica, N. Y., succeeded to the presidency; Dr. William C. Sandy, Harrisburg, Pa., was named president-elect, and Dr. Arthur H. Ruggles, Providence, R. I., secretary-treasurer.

"The Doctor" Now in a Permanent Home.

The $150,000 reproduction of the Sir Luke Fildes masterpiece "The Doctor" first shown by the Petrolagar Laboratories at Chicago's Century of Progress Exposition in 1933, was recently presented by its owners to the new Rosenwald Museum of Science and Industry in that city.

Following the two World's Fairs, "The Doctor" Exhibit went on a tour of 50,000 miles and was viewed by over five million people in eighteen principal cities throughout the country.

Designed to remind the public of the importance of the family physician, it required the full time of the late Chicago sculptor, John Paulding and the noted artist Rudolph Ingerle and a large corps of assistants, and took nearly a year to complete.

In its new location in the Rosenwald Museum it will be seen by millions of visitors annually.

Dr. F. E. Steere

Was elected a member of the town Council of Claremont at the elections in June.

Dr. Howard T. Holden,

Class of '34, University of Virginia, Department of Medicine, returned to the University Hospital on July 1, where he will be connected with the Eye, Ear, Nose and Throat Department for two years.

The American Gynecological Society

Held its annual meeting at Grove Park Inn, Asheville, N C., May 30, 31 and June 1, under the presidency of Dr. N. Sproat Heaney of Chicago. At the business session, Dr. Frederick C. Holden of New York City was elected president, and Dr. Richard W. Te Linde of Baltimore was re-elected secretary. This organization has a limited membership and Dr. M. P. Rucker of Richmond is the only member from this State.

Miss Helen Frances Ziegler,

Dean of the School of Nursing of the Medical College of Virginia, has accepted an appointment as dean of the School of Nursing at Vanderbilt Uni-

XUM

versity, Nashville, Tenn., and will assume her new duties on September 1. Miss Ziegler has been with the Medical College of Virginia for nine years.

Doctors Honored at Randolph-Macon College.

At the commencement exercises of Randolph-Macon College, Dr. M. Pierce Rucker, Richmond, was awarded the degree of doctor of laws.

Dr. Howard R. Masters, also of Richmond, was named a member of the advisory committee of the alumni association.

Dr. Oswald Weaver,

Recently of Charlottesville, has been appointed to the staff of the State Colony for Epileptics and Feebleminded, succeeding Dr. W. V. Kelly. He is a graduate of the medical school of the University of Virginia, class of '36.

Dr. John H. Miller,

Class of '32, Medical College of Virginia, formerly of Thomas, W. Va., recently completed a two-year resident internship at the New York Post-Graduate Hospital in New York City, and is now on a year's fellowship at the New York Vascular Clinic under Dr. Irving Wright.

Dr. J. Gordon Rennie,

Recently associated with Dr. M. E. Nuckols, in Richmond, has gone to Bluefield, W. Va., where, beginning July 1, he will be associated with Dr. Charles M. Scott at St. Luke's Hospital, in the practice of proctology and surgery.

New York Polyclinic Medical School and Hospital Announces Appointments.

Dr. Joseph F. McCarthy has been appointed professor of urology and attending urologist; Dr. Charles J. Imperatori, professor of otolaryngology and attending otolaryngologist; Dr. Joseph E. J. King, professor of neuro-surgery and attending neuro-surgeon; and Dr. Edward H. Donnon, professor of obstetrics and attending obstetrician.

Dr. Athey Ragan Lutz,

Formerly of Huntington, W. Va., announces the opening of his offices at 1044 Market Street, Parkersburg, W. Va., where his practice will be limited to orthopedic surgery and fractures. Dr. Lutz, class of '27, Medical College of Virginia, is a fellow of the American Academy of Orthopedic Surgery.

The Jefferson Medical College.

The One Hundred and Thirteenth Commencement of the College was held on June 3. The graduating class numbered 134, bringing the total number of graduates to 16,447. Thirty-four members of this class were commissioned as First Lieutenants in the Medical Section of the Officers' Reserve Corps.

The annual Alumni dinner was held on June 2, with 527 alumni in attendance, and Alumni Day and Ex-Internes' Day clinics were held on the 1 and 2.

The Graduating Class of 1938 presented a portrait of Dr. Brooke M. Anspach, Professor of Gynecology, to the College, and also a plague, commemorating the services of the late Dr. Burns.

Dr. J. Torrance Rugh, who, for twenty years, held the chair of Orthopedic Surgery, retired at the end of the present session, and was elected Emeritus Professor of Orthopedic Surgery in the College and Consulting Orthopedic Surgeon to the Hospital.

Surgical Club in Norfolk.

An informal surgical club was formed in Norfolk, this year, and on May 5, the first annual banquet was held at the Town Club.

The meetings are characterized by round table discussions, a different member heading the discussion at each of the meetings, which are held in the Norfolk County Medical Society Library at eight p. m., on the first Thursday of each month, from October to June, inclusive. The subjects for the past year have been: Significance and interpretation of symptoms in upper abdominal surgery by Dr. Robert DuVal Jones; Fracture problems by Dr. Allen S. Lloyd; Endocrines in gynecology by Dr. Eugene Lowenberg; Low cervical Caesarian section by Dr. Richard B. Nicholls; Esophageal stricture and cardiospasm by Drs C. C. Cooley and A. A. Burke; Drainage in abdominal surgery by Dr. R. D. Glasser; Healing of tissues by Dr. M. H. Todd; Cancer of the breast by Dr. N. F. Rodman; Congenital anomalies of the female genital tract by Dr. Horace Ashburn; and Surgery of the biliary tract with particular reference to drainage of the common duct by Dr. C. C. Smith.

The following are the officers of the Club: President, Dr. Charles Lupton; Vice-president, Dr. C. J. Devine; and Secretary-treasurer, Dr. Allen S. Lloyd. Other members are: Drs. Ashburn, Burke, Crosby, Doggett, Glasser, DuVal Jones, Lowenberg, Mat-

XUM

thews, Nicholls, Peake, Rodman, Rogers, C. C. Smith, Spigel, Todd, Eley, Strauss, Motyca, Angus Wilson, Wallace, M. S. Andrews, Duncan, Collins, Cooley, Byrd, and Morgan. The following are honorary members in this Club: Drs. C. J. Andrews, Charles Doughtie, Stanley Graves, Lomax Gwathmey, P. St. L. Moncure, Levi Old, R. L. Payne, Julian Rawls, Frank Smart, and Foy Vann.

Dr. J. E. Hamner,

Of Petersburg, has been named first vice-commander of Petersburg Post No. 2, American Legion, election of new officers being effective on July 22.

Drs. Michaux and Graham.

Dr. Stuart Michaux and Dr. A. Stephens Graham announce that they have entered a partnership for the practice of surgery and gynecology at Stuart Circle Hospital, Richmond.

Dr. Nathan P. Fitts,

Formerly of Strasburg, is now located in Swan Quarter, N. C., where he is assistant health officer to Dare-Hyde-Tyrrell-Washington District Health Department. He recently completed a year's residency at the Decatur and Macon County Hospital, Decatur, Ill., following which he attended a Public Health course at the University of North Carolina.

Dr. Roger L. Creekmur,

Richmond, has been named physician and surgeon of the Richmond Fire Department, succeeding Dr. Greer Baughman who recent resigned. He is a native of Richmond and has been engaged in the general practice of medicine and surgery since graduation from the Medical College of Virginia in 1921.

The Graduate Nurses' Association of Virginia

Held its thirty-eighth annual convention at the Chamberlin Hotel, Old Point Comfort, the latter part of May. Miss Charlotte Pfeiffer, Stuart Circle Hospital, Richmond, was elected president; Miss Eloise Lanford, Richmond, and Miss Myrtle M. Hollow, Charlottesville, vice-presidents; Miss Katherine Gary, Richmond, secretary; and Mrs. Jessie Wetzel Faris, Richmond, executive secretary-treasurer (re-elected).

Dr. Sidney Trattner,

Richmond, was recently elected vice-president of the Richmond First Club.

Dr. R. G. McAllister,

Class of '37, Medical College of Virginia, after completing an internship at St. Luke's Hospital, Richmond, will locate about the middle of July at Williamston, N. C., where he will be associated with Drs. J. S. Rhodes and J. A. Eason in general practice.

Captain John L. Waring,

Formerly of Danville and father of Dr. J. B. H. Waring of Wilmington, Ohio, former Virginian, attended finals of the Virginia Military Institute in June, where he received much attention as the oldest living graduate present, being of the class of 1870. After a visit with his daughter in Richmond, Captain Waring will attend the Union-Confederate assembly at Gettysburg before returning to Ohio.

For Sale—

Elliott Treatment Machine. Perfect condition. $100.00. Write "Machine", care the VIRGINIA MEDICAL MONTHLY. (*Adv.*)

Home for Convalescences.

A home for the care and treatment of chronic aged and convalescences has been opened at Arrington, Nelson County, Virginia, under the care of Dr. and Mrs. Fred M. Horsley. Information upon request. (*Adv.*)

Obituary Record

Dr. William Andrew Brumfield,

Prominent physician of Farmville, died of a heart attack on May 29. He was a native of Pittsylvania County and sixty-three years of age. He graduated from the University of Virginia, School of Medicine, in 1897. Dr. Brumfield was a widely known health authority and had served as director of the Southside Health District for the past twelve years. Before going to Farmville, he was health officer at the Virginia Polytechnic Institute. Dr. Brumfield had been a member of the Medical Society of Virginia for thirty-five years. His wife and seven children survive him.